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CONTENTS

	PAGE
Clinic of Drs. Charles A. Filott and Walter H. Nadler, <i>Wesley Memorial Hospital, Northwestern University Medical School Chicago</i>	
THE MANAGEMENT OF DIABETES MELLITUS ASSOCIATED WITH PULMONARY TUBERCULOSIS	1453
Clinic of Dr. Lewis J. Pollock, <i>Northwestern University Medical School</i>	
NEUROLOGIC DIAGNOSIS	1473
Clinic of Drs. Solomon Strouse and Phil A. Daly, <i>Chicago Lying In Hospital for North-western University</i>	
DIABETES AND PREGNANCY	1491
Clinic of Dr. Ralph C. Hamill, <i>Wesley Memorial Hospital</i>	
DISABILITY DAMAGES, OR DISEASE	1499
Clinic of Dr. Harold A. Bachmann, <i>From the Cardiac Clinic of the Children's Memorial Hospital</i>	
THE PREVENTION OF HEART DISEASE IN CHILDHOOD	1531
Clinic of Dr. William H. Holmes, <i>Wesley Memorial Hospital</i>	
CASES ILLUSTRATING THE SYMPTOMATOLOGY OF BRAIN-STEM LESIONS AND OF CHRONIC SUBDURAL HEMORRHAGE	1541
Clinic of Dr. Sidney Strauss, <i>Cook County and Michael Reese Hospitals</i>	
DISEASES OF THE BLOOD WITH SPECIAL REFERENCE TO HEMORRHAGES	1563
Clinic of Drs. Julius H. Hess and Philip Rosenblum, <i>Soroh Morris Children's Hospital of the Michael Reese Hospital</i>	
GASTRO-INTESTINAL HEMORRHAGE IN CHILDREN	1577
Clinic of Dr. Jacob Meyer, <i>Michael Reese Hospital</i>	
I ORTHOSTATIC ALBUMINURIA	1591
II ACUTE GLOMERULAR NEPHRITIS	1593
III CHRONIC GLOMERULAR NEPHRITIS	1595
IV ACUTE YELLOW ATROPHY	1598
Clinic of Dr. Harry A. Singer, <i>Cook County Hospital</i>	
SOME COMPLICATED AND UNCOMMON GASTRIC LESIONS	1601
Clinic of Drs. William A. Brant and E. P. Wolf-Billitzer, <i>Michael Reese Hospital</i>	
CLINICALLY DORMANT CARCINOMA AND THE IMPORTANCE OF A COMPLETE PHYSICAL EXAMINATION	1627
<hr/>	
Index to Volume 9	1637

THE MEDICAL CLINICS OF NORTH AMERICA

Volume 9

No 6

CLINIC OF DRs CHARLES A ELLIOTT AND
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THE MANAGEMENT OF DIABETES MELLITUS ASSOCIATED WITH PULMONARY TUBERCULOSIS

DIABETES mellitus and pulmonary tuberculosis were formerly regarded as an almost invariably fatal combination. Conflicting indications were encountered in the treatment of the two conditions, in that a high caloric intake was required in tuberculosis, whereas a diet restricted below the point of individual tolerance was necessary in diabetes. The use of insulin has made it possible to meet both requirements. By means of a balanced diet containing a relatively large amount of fat in safe proportion to the available glucose enough calories can be furnished to maintain the patient's weight, and by means of sufficient insulin the endogenous supply can be augmented to insure the utilization of the carbohydrate given. As a result of the better management of diabetes which has thus been made possible, the prognosis of cases complicated with tuberculosis has been greatly improved.

On the part of the patient sufficient intelligence to be able to follow a strict weighed diet and a willingness to co-operate are prerequisite to successful management. On the part of the physician continued observation, in order to determine the efficacy of control, is necessary. As to what constitutes effective control of diabetes there is some difference of opinion, and, consequently, various criteria are followed. Most writers on

the subject have aimed to prevent glycosuria, although some permit the excretion of occasional or slight amounts of sugar. Others have aimed to prevent hyperglycemia. Whether it be true, as statistics indicate, or untrue that tuberculosis is no more common in diabetics than in the general population, it seems obvious that in diabetes with tuberculosis a normal glycemia is desirable. Persistent hyperglycemia, which predisposes to infections in general, must predispose to the extension of latent or active tuberculous foci by providing a medium favorable to the growth of the tubercle bacilli. Control of glycemia seems the ideal goal of treatment in the light of our present knowledge.

The results of urine examinations do not furnish information as to the blood-sugar level. Although it is true, especially in the milder cases of diabetes, that the blood-sugar often becomes normal when glycosuria has been produced, hyperglycemia may exist without glycosuria. That frequent blood-sugar estimations seem impractical and may be difficult to obtain is no argument against their value. Many of the objections to frequent determinations have been overcome by the introduction of accurate micro methods. The use of effective preservatives promises to make these examinations generally practicable¹. In our experience a knowledge of the fasting blood-sugar has been indispensable. We have used, for the most part, specimens of capillary blood which the patient has been taught to collect from his fingertip. For this purpose he uses a spring lancet which makes a small incision from which about 10 drops (about 0.2 c.c.) of blood are caught in a small vial containing 5 milligrams of Sander's² preservative, a mixture of sodium flourid and thymol. The blood does not coagulate, glycolysis is prevented for several days by the thymol. For sugar determinations of the micro samples the Randles-Grigg³ modification of the Folin-Wu method has been used with an accuracy well within clinical requirements.

We wish to present 4 patients with severe diabetes and advanced tuberculosis. In one case the tuberculosis has apparently healed, in another the process has become quiescent, in the third patient conditions favorable to healing have been brought about and a good prognosis is justifiable, in the last case, however,

although aglycosuria has been maintained, the fasting blood-sugar level remains above normal, progress has been very slow, and the outlook is grave. Frequent adjustments in insulin dosage were required before it was possible finally to establish an equilibrium between the total insulin available and the glucose intake. The amount of insulin required during fever and obvious tuberculosis toxemia has been, in several cases, extraordinarily large. A study of the blood-sugar variations occurring at intervals during the day has been of great help in placing the larger doses in proper time relation to the peaks of hyperglycemia. By means of these diurnal curves it has been possible to adjust the insulin dosage so as to avoid hypoglycemic reactions. As a result of the endeavor to give the patient sufficient insulin hypoglycemic reactions frequently occurred as his sugar tolerance increased. Such reactions were easily relieved. This slight danger is counterbalanced by the improvement which follows when normal glycemia is maintained. As secondary therapeutic measures bed rest, fresh air, and sunlight are important.

CASE I

Reverend C. C., thirty-eight years of age, was admitted to Wesley Hospital for instruction in regard to diet on September 14, 1922. Glycosuria had been discovered some six months earlier by a physician who had been consulted because of polyuria and loss of weight and strength. After following a restricted diet only occasional traces of sugar appeared. He had never weighed more than 140 pounds, which is below normal for his height of 69 inches. Of previous illnesses, only measles and scarlet fever in childhood, and an operation in 1919 for subacute appendicitis is recalled. There is no family history of diabetes. A sister died of pulmonary tuberculosis at the age of thirty-one years.

On examination the patient appeared extremely thin. Weight 121 pounds (55 kgm.). No fever or cough. General adenopathy was present. Teeth and tonsils showed no infection. Heart, lungs, and abdomen were negative. Blood-pressure normal. Roentgen examination of the chest was not made. No tubercle bacilli were found in the specimens of sputum obtained. Was-

sermann test negative Metabolic rate -9 per cent The first twenty-four-hour specimen of urine contained 5 gm of glucose After two days on a low carbohydrate diet the urine was sugar free He was discharged after a week with a blood-sugar of 0 094 per cent on a diet of Ch 60, P. 80, F. 120 (calories 1640) During the next month he remained aglycosuric The diet was then increased to Ch 90, P 90, F 150 (calories 2020), the fasting blood-sugar was 0 124 per cent About six weeks after returning to his work in another city he caught cold and glycosuria reappeared He spent a week in bed with grip and bronchitis

Readmitted to the hospital on January 2, 1923, he appeared pale and emaciated Weight 117 pounds (53 kgm) Fever, cough, and glycosuria were present. Blood-sugar 0 224 per cent Dulness, moist râles, and muffled breath sounds were detected in the apex of the right lung Many tubercle bacilli were found in the sputum Stereoscopic radiograms revealed extensive changes in the upper right lobe and in both hilum regions, together with slight change in the left apex. The lung markings were interpreted as chronic, with activity in the right upper lobe

Further Clinical Course—Glycosuria disappeared promptly with bed rest and a diet which met basal requirements The fasting blood-sugar after five days decreased from 0 224 to 0 150 per cent The temperature was normal after ten days The use of iletin was begun on January 8, 1923, in doses of 5 units three times daily The diet, and particularly the carbohydrate supply, was gradually increased without reappearance of glycosuria or further elevation of the blood-sugar level On February 8th, after several days on Ch 130, P 100, F 200 (calories 2720), the fasting blood contained 0 163 per cent of sugar He had gained 6 pounds in weight On February 28th, five days after a further increase of carbohydrates to 155 gm , the urine contained a large amount of sugar The insulin dosage had remained at 15 units daily After a day of fasting the blood-sugar was 0 144 per cent , but with a return to a diet containing 140 gm of carbodyhrate and 218 gm of available glucose it rose to 0 337 per cent The dietary management described is open to criticism in the light of present practice, but it was feared at that time to increase the



CASE I
(c.c.)
S. 22 25

Fig 251.—Chest radiogram of Case I, August 22, 1925. Clinical diagnosis Arrested tuberculosis, diabetes under control. The right lung shows dense shadow bands in the upper field extending from the lung root to the periphery. Within the dense shadow are small circumscribed areas of lessened density. The entire upper third presents lighter shadows suggestive of a recent process. There is evidence of pleural thickening and retraction over the right apex. The left lung field is quite clear except for some haziness in the apex. The lung roots are markedly increased, bronchial markings extend well out to the periphery in all directions.

sists greatly in order to supply the desired number of calories. It is evident that the successive additions in carbohydrate passed

the point of tolerance. Such additions might have been utilized by sufficient iletin, but iletin was a new remedy, and we were fearful of producing hypoglycemic reactions.

Soon after this the patient went to a tuberculosis sanitarium in New Mexico. Great difficulty was experienced at first in controlling glycosuria, but finally, with a reduction of diet to Ch 75, P 65, F 150 (1910 calories and 127 gm of available glucose) and with an increase of insulin to 30 units daily, he became and remained sugar free. After a year he was discharged as an arrested case of pulmonary tuberculosis. He has gradually

Date, 1925	Diet					Weight (kgm)	Urine sugar	Blood sugar	Insulin
	Ch	P	F	Total glucose	Calories				
1/19	75	65	150	128	1910	60	0	0 155	10-10-10
1/26								0 129	
2/2								0 190	
2/9								0 112	
2/16								0 132	
2/23								0 135	
3/9	90	75	160	149	2100			0 148	
3/16	90	75	180	151	2280			0 167	
3/23								0 167	
3/30								0 157	
4/27								0 182	
5/11	80	75	180	141	2210			0 117	15-10-10
5/18	75	75	210	139	2190			0 120	
6/13	75	65	210	135	2450		+	0 184	
7/11	75	70	210	136	2170			0 110	
7/20								0 107	
7/25								0 095	
8/4								0 131	15- 0-15

increased his activity and is about to resume his work. He feels and looks well and has no fever or cough. Weight 130 pounds (60 kgm). Blood-pressure and urine are normal. The peripheral vessels are soft. Radiograms show an old healed process with cavity formation in the right upper lobe (Fig 251). Whether other markings suggestive of a recent process denote present

activity cannot be determined by Roentgen examination There are no clinical signs of active tuberculosis

Since January of this year blood-sugar estimations have been made usually at weekly intervals Until June, when he returned to this city, specimens of blood were drawn by the patient and mailed from New Mexico The specimens were apparently efficiently preserved by a mixture of sodium fluorid and thymol The blood-sugar levels and the diet and insulin régime followed are shown in the table on page 1458

Recently the insulin dosage has been reduced to 15-0-10 because of mild hypoglycemic reactions occurring in the evening On August 22, 1925 a series of blood-sugar estimations was made at intervals during twenty-four hours in order to determine whether the insulin dose was proper in amount and in time The following results were obtained

	Percent
Fasting blood-sugar	0 125
1½ hours after 15 units insulin and breakfast	0 126
Before noon meal	0 070
1½ hours after lunch	0 101
Before 10 units insulin and evening meal	0 082
1½ hours after evening meal	0 176
3½ hours after evening meal	0 170
Following morning (fasting)	0 092

Since capillary (arterial) bloods were examined, the readings obtained shortly after meals and insulin probably represent values higher than would have been obtained with corresponding venous blood

Discussion—This patient has diabetes mellitus and pulmonary tuberculosis which has been arrested or has been healed That the tuberculosis was chronic and dated probably from childhood is indicated by Roentgen examination and by a history of exposure Glycosuria appeared in the spring of 1922, and signs of active tuberculosis in December of the same year The early management of the diabetes, based on the maintenance of aglycosuria, was inadequate A reluctance to increase fats and to administer insulin in large amounts were handicaps Later, after sufficient insulin was supplied, healing of the tuberculous

lesions was accelerated It is of interest that this patient, who has been taking insulin daily since January 8, 1923, has suffered little inconvenience and no apparent harmful effect Prolonged observation of the blood-sugar indicates that the diabetes is under control

CASE II

Mr C R , an electrician, thirty-five years of age, entered the hospital November 12, 1924 He had been ill for six weeks, but had felt below par for four years, following a series of carbuncles, during which he lost 60 pounds in weight He had had frequent head and chest colds For a year increased thirst and urination (nocturia four or five times) had been present, but no urine examinations were made

Six weeks before admission he became chilly and then febrile on returning from work Grip was diagnosed After four days in bed he resumed his duties, although he did not feel well A week later he had a severe chill and his temperature rose to 104° F Sharp pains in his left chest and a severe, productive cough appeared, the sputum was not blood-tinged A diagnosis of pneumonia was made After a week he was able to sit up, but cough, dyspnea, hoarseness, and night-sweats continued He states that during this time he drank more than a gallon of water a day There is no family history of tuberculosis Both parents died of diabetes and apoplexy Two brothers and 8 sisters enjoy good health

Examination showed a fairly well-nourished man, weight 145 pounds (66 kg), height, 64 $\frac{1}{2}$ inches Fever, cough, and hoarseness were present Extensive pyorrhea, with deep pockets and considerable pus, and slight, chronic tonsillar infection were found The laryngeal mucosa was inflamed, an ulceration on the posterior wall between the arytenoids was suggestive of tuberculosis Thyroid normal Slight general adenopathy Scars of incised carbuncles remained on the back of the neck The left chest lagged on inspiration, posteriorly, the entire left side was dull to percussion, a friction-rub and many rales were heard over the upper half The right chest was normal Heart findings were negative save for tachycardia Abdomen normal Blood-

pressure and reflexes normal. The Wassermann test was negative. The blood-count was normal. The urine contained a large amount



Fig 252.—Chest radiogram of Case II, November 12, 1924. The upper half of the left lung shows diffuse markings suggestive of an active inflammatory process. Except at the apex most of the peripheral portion is clear, intermediate and hilum structures are greatly increased in tissue density. Certain decreases suggest cavitation with indistinct walls. The right lung field is normal throughout.

of sugar and a trace of albumin. Fasting blood-sugar 0.282 per cent. Many tubercle bacilli were found in the sputum. Roent-

gen examination showed an unusual shadow arrangement in the right lung suggestive of very active interstitial inflammation, most of the peripheral portion was clear except at the apex, the intermediate and hilum structures were greatly increased in density (Fig. 252).

Clinical Course.—After a week on a diet of 1720 calories containing 106 gm of available glucose (Ch 60, P 55, F 140) and 30 units of insulin daily the blood-sugar level dropped from 0.280 to 0.185 per cent and the urine became sugar free. With a slight increase in diet 45 units of insulin were required to keep the fasting blood-sugar at a normal level. On January 5th the food intake was increased to Ch 85, P 65, F 200 (calories 2400 containing 143 gm of available glucose), thus diet has since been followed. The insulin required for the maintenance of normal glycemia has gradually decreased from 70 to 45 units. Thus, during February, 70 units were given, during March, 60 units, during April, 50 units, and since that time 45 units daily. At first administered in three doses, the insulin has (since May) been given in a morning dose of 25 and an evening dose of 20 units. A further reduction seems to be indicated at the present time. Only a few slight reactions have been experienced. The clinical course, viewed from the standpoint of the blood-sugar, is illustrated in Fig. 253. The decreasing insulin dosage required to maintain normal glycemia indicates an increasing sugar tolerance.

The urine has remained sugar free. Occasional afternoon rises in temperature persisted until January 28th. On returning to his home on February 21st the patient's cough had disappeared, and the ulceration of the larynx had almost healed, sweating and tachycardia were his only symptoms. Only a few rales were heard over the left upper lobe, dulness and diminished breath sounds persisted. The patient continued to rest at home until August 1st. Since then he has been at work. His duties, however, require little physical exertion. The tuberculous process seems to be quiescent and the diabetes to be under control.

Discussion.—From the history of this patient it appears that diabetes has been present for over four years. Obesity may have

been a predisposing factor. The carbuncles which developed were probably complications associated with hyperglycemia. Distinctive symptoms of diabetes existed for over a year before the onset of tuberculosis in pneumonic form. The favorable course is due to the co-operation of the patient, who has weighed his diet and taken his insulin with scrupulous care. The frequent

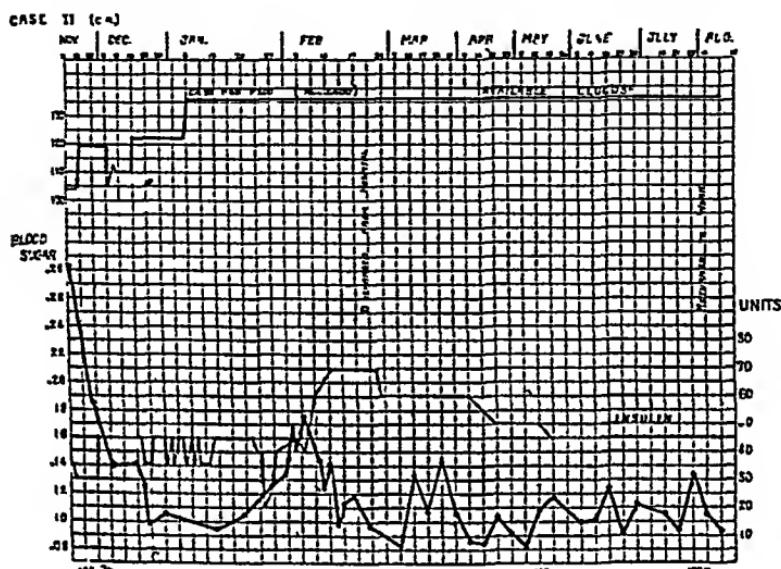


Fig. 253.—This chart represents the clinical course of Case II from the standpoint of diabetic management. The upper line shows the grams of glucose available in the diet, the heavy line, the fasting blood-sugar values, and the lighter, dotted line the insulin dosage. A gain of tolerance is indicated.

blood-sugar examinations have been of great aid to us in regulating the insulin dosage, to the patient they have been of great disciplinary value. The decreasing amounts of insulin required to maintain a normal fasting blood-sugar level indicate a gain in sugar tolerance.

CASE III

Mr J B, age forty-nine years, was referred for examination on January 17, 1925. He had been actively engaged in business in spite of various symptoms to be described. Glycosuria had

been discovered ten years previously in the course of an examination for life insurance. At that time he weighed 210 pounds.



Fig 254.—Chest radiogram of Case III, January 22, 1925. The lung apices are clear. On the left side from the first to the third interspace markings extend from the lung root to the periphery, in the outer portion there is an annular shadow with thick walls, this is evidently a cavity without fluid content. Pleural thickening is confined to the region described. Both lung roots show shadow increase and large amount of calcification.

With some attention to diet he lost 20 pounds. In the few examinations of the urine made since then traces of sugar were

found, polyuria was intermittent. In the past year he had lost 22 pounds. For six months a burning pain in the shoulder and beneath the sternum had been present, and for about the same length of time he had had a morning cough. There is no family history of tuberculosis or diabetes.

Examination showed a short, thick set, flabby man, 64 inches tall, and weighing 160 pounds (73 kg.). Temperature 99° F. There were signs of infection about the teeth. Heart normal.

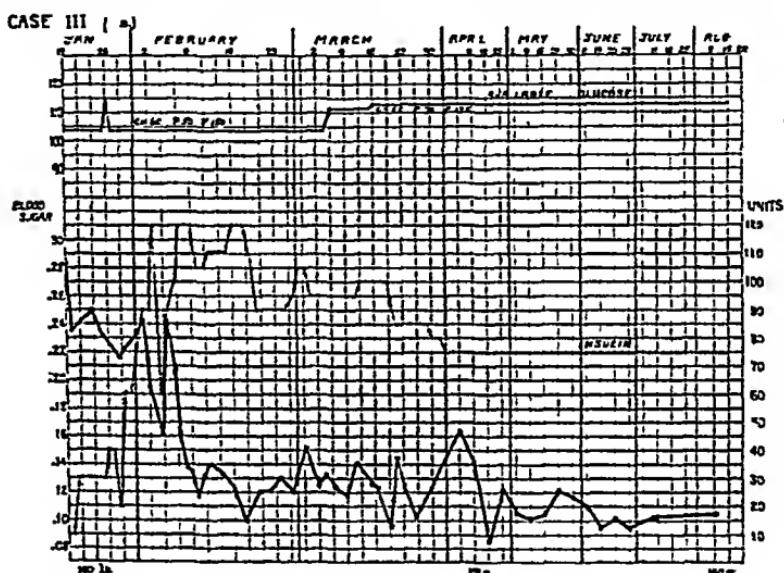


Fig. 255.—The fasting blood-sugar values of Case III during seven months of treatment are shown. The number of insulin units administered daily is indicated by means of the lighter dotted line. Normal glycemia has been maintained.

except for increase in rate (92). Blood-pressure 140/88. Many râles were heard over the left lung. The Wassermann test was negative. The urine contained sugar and diacetic acid, no albumin or casts. Blood-sugar 0.282 per cent. Tubercle bacilli were found in the sputum. Roentgen examination showed evidence of an old healed tuberculosis of the lung roots, markings in the left upper lobe suggested the probability of breaking down of one or more of these foci, a single, fairly large cavity was made out (Fig. 254).

Clinical Course.—As indicated in Fig. 255, a diet containing 104 gm of available glucose and 30 units of insulin daily had but little effect on the fasting blood-sugar. An increase of insulin to 120 units daily was required to produce a normal glycemia. Gradual reductions in the insulin dosage were subsequently possible. A few days after a reduction to 85 units peculiar reactions characterized by disturbed vision and symptoms resembling hysteria occurred. In order to study the effect of the insulin and to

CASE III

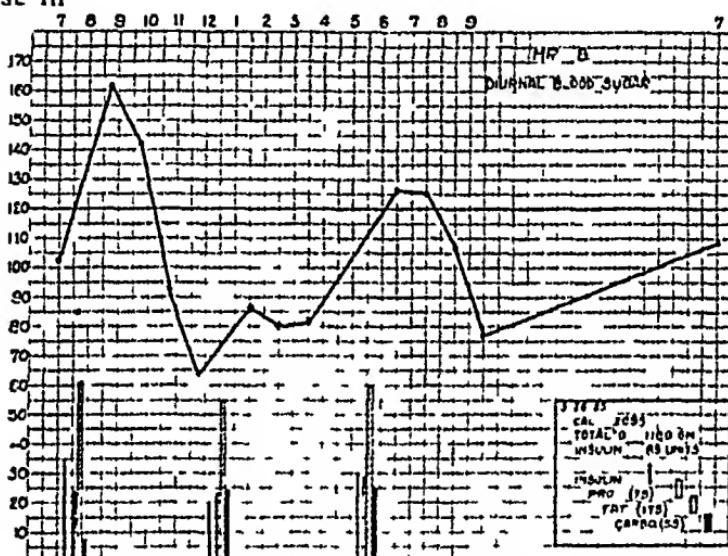


Fig. 256.—Diurnal blood sugar curve of Case III, March 26, 1925. The diet of 2095 calories contained 117 gm of available glucose. Breakfast which contained approximately 27 gm of available glucose, was preceded by 35 units of insulin, the noon meal, with 42 gm, by 20 units and the evening meal, containing 47 gm, by 30 units of insulin. The maximum rise (0.162 per cent) occurred after breakfast. The lowest blood sugar level (0.061 per cent) was found before lunch at a time which corresponded with the onset of hypoglycemic symptoms. The fasting levels were normal. After a slight reduction of the morning dose of insulin to 30 units the reactions disappeared.

determine the proper division of the dose, blood-sugar estimations were made at intervals during twenty-four hours (Fig. 256). With the insulin division of 35-20-30 the peak of the sugar curve occurred after breakfast, before luncheon, at the time of the

reaction described, a low point of 0.064 per cent was reached, a small rise followed the evening meal. A curve of this character was found by Jonas and his co-workers⁴ to be the usual one obtained under similar conditions. Since the estimations were made with samples of capillary blood it is probable that all except the fasting specimens contained more glucose than would have been present in corresponding venous samples. On April 1st, when the patient was discharged from the hospital, 75 units of insulin were required for the utilization of a diet which had been increased from 104 to 113 gm in available glucose content. During the period of hospitalization his afternoon temperature rose to 99° or 100° F. Since returning to his home in Georgia the patient has mailed samples of blood for examination. The urine has remained sugar free and normal glycemia has been maintained. Neuritic pains in the shoulders, rise in temperature to 99.2° F., and slight cough are the only symptoms noted.

Discussion—This patient has had diabetes for at least ten years. The condition, practically disregarded, became severe. The time of onset of the tuberculosis is unknown, a healed or latent process probably existed for years. Signs of activity had been present for about six months. At the beginning of treatment 120 units of insulin were required to maintain normal glycemia, after several months 75 units were sufficient. Rest and other measures of a tuberculous régime are being carried out. A favorable prognosis seems justifiable.

CASE IV

Mr J C, a foreman of auto repairs, thirty-three years of age, was admitted in January, 1924. Four months previously he had noticed a dryness of his throat, unusual thirst, and polyuria. He had quickly lost in weight from 155 to 120 pounds. His past history is negative except for illnesses of childhood, an appendectomy in 1915, and rather frequent colds. There is no family history of diabetes or of tuberculosis.

Examination showed a tall, thin man of ruddy complexion. Height 70 inches, weight 55 kg. No fever or cough. Teeth and tonsils were free from infection. Cervical glands were palpable.

Cardiovascular system normal No abnormal lung findings were detected Abdomen negative Reflexes normal Wassermann test negative Metabolic rate -3 per cent The twenty-four-hour urine contained 41 gm of glucose, no diacetic acid, albumin, or casts Blood-sugar 0 416 per cent

Clinical Course —After two weeks of instruction, during which he had gained six pounds, the patient was discharged, sugar free, on a diet of 2160 calories containing 130 gm of available glucose and on an insulin dosage of 14 units daily On returning to work slight intermittent glycosuria appeared Insulin was gradually increased to 30 units daily After several months his regular visits ceased In December, 1924 he reported that he felt well, but that intermittent glycosuria continued From January to April, 1925 a series of blood-sugar determinations was made, with the following results 0 392, 0 200, 0 351, 0 322, 0 323, 0 250, 0 209, 0 308, 0 257, and 0 202 per cent An increase in insulin to 45 units during February and to 50 units during March failed to produce normal fasting levels In April he was persuaded to return to the hospital for the adjustment of the insulin dosage The results of six days of observation were as follows

Date 1925	Diet			Calories	Available glucose	Urine	Blood sugar	Insulin
	C	P	F					
4/6	90	60	210	2490	146	+	0 315	0-15-20
4/7	90	60	210	2490	146	tr	0 256	25-15-20
4/8	90	60	210	2490	146	tr	0 220	25-15-20
4/9	90	60	210	2490	146	tr	0 250	25-15-20
4/10	90	60	210	2490	146	tr	0 146	30- 0-25
4/11	90	60	210	2490	146	0	0 184	30- 0-25

After leaving the hospital he refused to return for observation Despite warnings he finally disregarded his diet and stopped insulin Immediately he began to feel badly, and on June 4th he was sent to a hospital in a semicomatose condition

On readmission to Wesley Hospital on July 8th he complained of a severe and very productive cough which had come on without

any evidence of a cold. He had lost 30 pounds in two months. His appetite was good. Extreme thirst had been present for

CASE IV (c)

7 1+ 25



Fig 257.—Chest radiogram of Case IV. On the left side there is a dense shadow in the upper half of the lung; the pleura is retracted, cavitation and interlobar pleural changes are indicated, the costophrenic angle is partially obliterated and adhesions of the diaphragm limit excursion. On both sides bronchial increases are marked, especially in the lung roots. The right lung is otherwise normal.

two weeks. He was very thin and pale. Fever was present, but he had had no night-sweats. There was a marked dulness over

the upper two-thirds of the left chest, tactile and vocal fremitus were increased and many râles were heard over the dull area. Many tubercle bacilli were found in the sputum. The urine contained a large amount of glucose. Blood-sugar 0.378 per cent. Chest radiograms showed advanced and active tuberculosis involving the upper left lung (Fig. 257).

Recent Course.—The recent course is indicated in the following table.

Date, 1925	Diet					Urine	Weight (kg.)	Blood sugar	Insulin
	Ch	P	F	Calories	Available glucose				
7/10	100	90	75	1435	160			53	0.378, 15-15-15
7/11	100	90	150	2110	167				25-25-25
7/13	100	90	150					0.215	25-25-25
7/14	100	90	175	2335	170				35-35-35
7/16	100	90	175					0.236	35-35-35
7/19	100	90	175					0.250	25-25-10
7/20	100	90	175					0.333	30-30-20
7/22	100	90	175					0.274	30-30-20
7/25	100	90	175					0.238	30-30-20
8/1	100	90	175					0.174	30-30-20
8/3	100	90	175					0.266	30-30-20
8/11	100	90	175				51	0.199	30-30-20
8/13	100	90	175					0.274	35-20-25
8/14	100	90	175					0.267	35-20-15
8/16	90	70	220	2620	152				35-10-25
8/17	90	70	220					0.204	35-10-25
8/18	90	70	220					0.294	35-10-25
8/19	90	70	220					0.257	35-10-25
8/20	90	70	220					0.260	35-10-25
8/22	90	70	220						35-10-30
8/23	90	70	220					0.210	40-10-30
8/26	90	70	220					0.180	40-10-30
8/27	90	70	220						40-15-25
8/28	90	70	220					0.238	40-10-25
9/1	90	70	220					0.184	45-10-30
9/2	90	70	220					0.211	45-10-30
9/3	90	70	220					0.222	45-10-30
9/4	90	70	220				50		45-15-30
9/5	90	70	220					0.198	45-15-30

Afternoon fever (99° to $101\frac{4}{5}^{\circ}$ F) continued until August 8th, since then there has been only an occasional slight rise in temperature. Cough and expectoration have decreased and tubercle bacilli are difficult to find. The upper left chest remains dull, but fewer râles are heard. A weight of 110 pounds has been maintained. His appetite is fairly good. Glycosuria disappeared after a few days but, as shown in the above table, hyperglycemia has persisted. The amount of insulin has been gradually increased to 90 units, which causes an occasional slight reaction. An all-day curve showed a marked elevation in blood-sugar after breakfast and approximately normal values during the rest of the day, during the night, however, the sugar rose to the fasting levels indicated in the table. Despite the high fasting blood-sugars and the marked rise after the morning meal the twenty-four-hour specimens of urine preserved with toluol have failed to show glucose with Benedict's solution. Quantitative estimations using the Folin-Berglund method, have given normal results. For example, on August 17th, when the fasting blood-sugar was 0.204 per cent., the twenty-four-hour urine contained only 0.34 gm. on August 25th, with a blood-sugar of 0.189 per cent., the urine contained 0.40 gm., on August 27th, with 0.210 per cent. blood-sugar, there was 0.335 gm. and on September 2d a blood-content of 0.211 per cent., was contrasted with a urine sugar of 0.493 gm.

Discussion.—This patient probably had an unrecognized latent tuberculosis activated by a severe diabetes. Initial treatment directed against the appearance of glycosuria was not successful. Continued hyperglycemia favored the extension of the tuberculosis. The danger of coma from the neglect of diet and insulin is illustrated. That hyperglycemia may exist unaccompanied by glycosuria is also shown. It has not, as yet been possible to maintain normal glycemia throughout the twenty-four hours, the blood-sugar rises during the night to the high fasting levels recorded. By means of a night dose of insulin and a readjustment of diet, using diurnal curves as a guide, we hope to be able to prevent hyperglycemia. Healing of the tuberculous process seems to have been retarded by our failure to establish a normal blood-sugar level.

CONCLUSION

The cases presented of severe diabetes and advanced tuberculosis illustrate the possibility and the advantage of maintaining the blood-sugar at or near a normal level. Normal glycemia, clinically, favors the healing of the tuberculous lesions, hyperglycemia predisposes to their extension.

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CLINIC OF LEWIS J POLLOCK

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NEUROLOGIC DIAGNOSIS

In beginning the clinics of the following year I have the privilege of addressing you upon the subject of neurology. For generations it has been traditional that neurology is the most difficult and, at times, I fear, useless subject taught in the medical curriculum. This misconception is in my opinion, due largely to the manner in which neurology has been taught and learned. I believe that neurology presents itself in so logical a form that with a certain foundation it becomes one of the easiest subjects which you are called upon to study.

I am quite in agreement with the students who state that it is impossible to learn the symptoms, signs, and pathology of the various diseases of the nervous system by memory. I believe it quite useless to attempt to remember the signs and symptoms of meralgia paresthetica, of syringomyelia, or what-not, as such. I believe it will be found to be exceedingly difficult and, at times, impossible to remember those signs and symptoms for a time even long enough to pass an examination. If, however, we approach the subject of neurology from the standpoint of anatomy and physiology the matter is considerably simplified. If we but localize certain pathologic lesions in the central nervous system, we find there are but few diseases which could so affect the nervous system at that particular locality.

For example, if by means of the signs and symptoms of a particular case we localize the lesion about the central canal of the spinal cord, there are but two conditions which might logically cause this pathology—syringomyelia and an intramedullary tumor. Instead, therefore, of having to remember the signs and

symptoms of many diseases, it would be necessary only for us to know some of the underlying anatomic and physiologic facts which would enable us to so localize the lesion.

Much of the difficulty which exists in learning neurology I believe is due to the method of learning the anatomy and physiology of the nervous system. I do not believe it to be essential to be able to trace the fibers of the inferior longitudinal fasciculus, or to describe any obscure formation within the nervous system. Much of the elementary training in these subjects is offered as

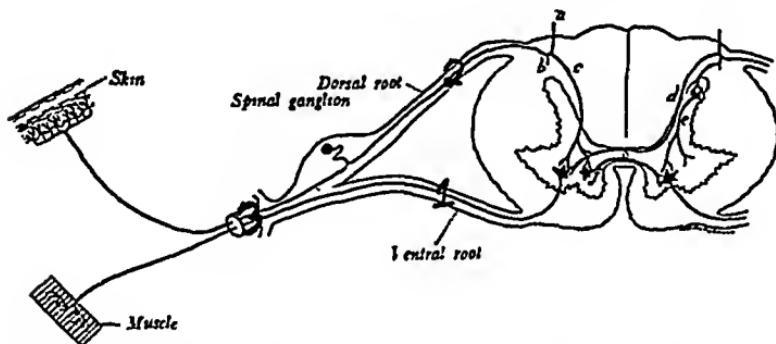


Fig. 258.—Diagrammatic section through the spinal cord and a spinal nerve to illustrate a simple reflex arc. *a*, *b*, *c*, and *d*, Branches of sensory fibers of the dorsal roots; *e*, association neuron; *f*, commissural neuron. (After Ranson.) A lesion at 1 produces flaccid paralysis, absence of deep reflexes, reaction of degeneration (Anterior root neuritis). A lesion at 2 produces pain, loss of deep reflexes, loss of all sensation (Compression by tumor, etc.). A lesion at 3 produces flaccid paralysis, absence of deep reflexes, reaction of degeneration, and loss of sensation (Peripheral neuritis).

a means of teaching a student how to think and where to find things to think about. It is necessary to know a few of the long fiber tracts, some of their connections or levels of reflexes, the levels of origin of cranial and spinal nerves, and the general conformation of the nervous system. This knowledge may be readily acquired.

Let us begin by visualizing a cross-section of the spinal cord. We find that it consists of white and gray matter, the latter in the form of a letter H. In the anterior part of the gray matter there exist a number of horn cells, known as anterior horn cells, from which originate the anterior root fibers. In the posterior

horn the posterior root fibers enter and connect with the anterior horn, constituting a lower reflex arc. If this arc is interrupted at any point the reflex which this particular arc subserves is destroyed (Fig. 258).

For example, if we interrupt this arc at the level of the second lumbar segment the knee-jerk disappears. This lower reflex arc is controlled by impulses from the cerebrum through the pyramidal tract. These impulses are of an inhibitory character, so that if we have a lesion of the pyramidal tract above the level of any deep reflex arc, that reflex is increased. We are already provided with a method for differentiating upper motor neuron lesions, or those occurring in the motor tract from the cortex, to the anterior horn cell in the spinal cord from those of the lower motor neuron, or that part of the motor system from the anterior horns to the motor end-plate in a muscle. In the former the deep reflexes are increased, in the latter they are diminished or absent. If the lesion is on the motor side of the lower reflex arc, we know that certain trophic changes occur whether the lesion is in the anterior gray matter of the cord, in the root, or in the peripheral nerve.

The most important trophic changes are a degeneration in muscle and nerve, as shown by an electric reaction of degeneration and atrophy of the muscles.

Tone which is a quality of muscles in contraction, is dependent upon an intact reflex arc, so if this arc is interrupted at any point flaccidity ensues.

Let us now visualize a few of the important tracts. We will observe that in the lateral region of the spinal cord the crossed pyramidal tracts ascend. Somewhat adjacent to them are the spinothalamic tracts. In the posterior columns of the cord ascend, uncrossed, the sensations for joint, muscle, and bone sensibility. Touch likewise chiefly ascends in these columns. The fibers subserving pain and temperature senses enter through the posterior roots and, crossing the gray matter of the spinal cord, ascend in the opposite spinothalamic tract (Fig. 259).

A lesion affecting the pyramidal tracts will produce a paralysis. Insomuch as this paralysis is of the upper motor neuron

type, it will produce an increase of the deep reflexes. Just as the deep reflexes are increased, so, as the result of diminished inhibition, tone is increased and spasticity is found in the paralyzed muscles. The separation of the lower part of the central nervous system from the upper levels releases certain protective

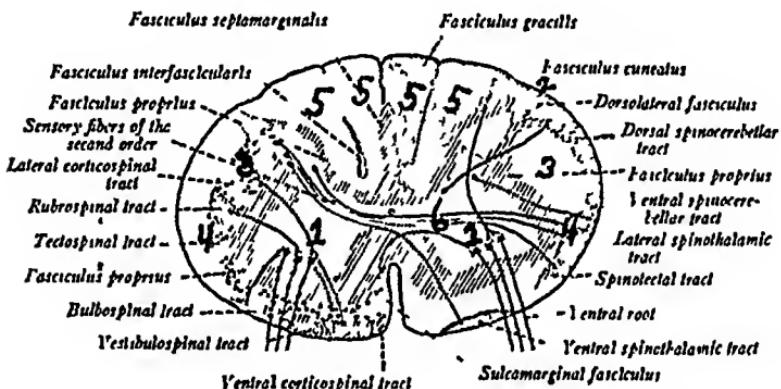


Fig. 259.—Diagram showing the location of the principal fiber tracts in the spinal cord of man (After Ranson). 1, Anterior horn; 2, posterior root; 3, pyramidal tract; 4, spinotriangular tract; 5, posterior columns (Goll's and Burdach's); 6, fibers to opposite spinothalamic tract. A lesion in 3 produces spastic paralysis, increased deep reflexes, and Babinski sign (Lrb's spinal spastic paralysis). A lesion in 1 produces flaccid paralysis, loss of deep reflexes, atrophy, and reaction of degeneration (Anterior poliomyelitis, progressive muscular atrophy). A lesion in 1 and 3 produces flaccid paralysis, absence of deep reflexes, atrophy and reaction of degeneration at the level of the involvement of the anterior horn, and a spastic paralysis with increased deep reflexes and Babinski sign below the level (Amyotrophic lateral sclerosis). A lesion in 5 and 2 produces absence of deep reflexes, pain, areas of sensory loss to touch and pain, loss of deep sensation, and sensory ataxia (Tonus dorsalis). A lesion in 6, 1, and 3 produces a picture similar to that described in a lesion of 1 and 3, along with a loss of pain and temperature sense in the levels where the central gray (6) is involved (Syringomyelia and, rarely, intramedullary tumor).

and other reflexes which consist of integrated motor movements, of which that combination known as a Babinski sign is one.

If, therefore, we see a case in which we have a spastic paralysis of both legs, with increased deep reflexes and bilateral Babinski with no sensory changes and no trophic disturbances, the condition can be due to only a bilateral involvement of the

pyramidal tracts and is produced by only a few diseases—a primary spinal spastic paralysis, or Erb's paralysis, or, rarely, it may be the beginning of some other disease (Fig. 260)

A lesion which destroys the anterior gray matter of the spinal cord produces a paralysis which, because the lower reflex arc is

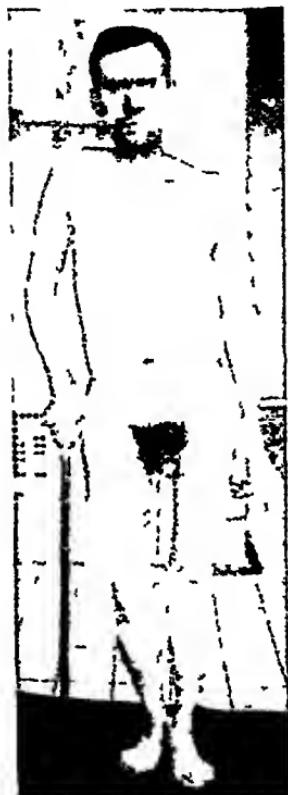


Fig. 260—A case of Erb's spinal spastic paralysis, showing spastic paraparesis of slow onset with increased deep reflexes and bilateral Babinski sign

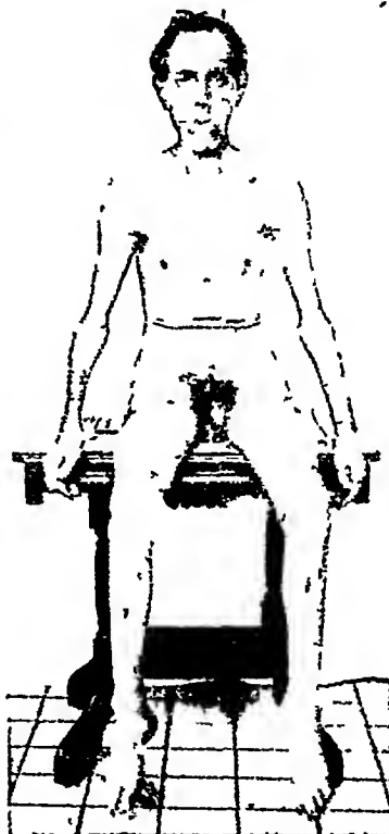


Fig. 261—A case of progressive muscular atrophy

interrupted, is associated with an absence of deep reflexes. The trophic supply to the muscles having been destroyed, it is likewise associated with a reaction of degeneration and an atrophy of the muscles

If, therefore, we have a lesion which produces a flaccid paralysis with absence of deep reflexes, atrophy and reaction of degeneration and no sensory changes, the lesion must be either in the anterior roots or in the anterior horn, usually in the latter, because there are very few conditions in which the roots themselves may be involved. If the lesion resides in the anterior horn, it may be due to an anterior poliomyelitis which, when acute, is the ordinary infantile paralysis and, when chronic, is a progressive spinal muscular atrophy (Fig. 261)

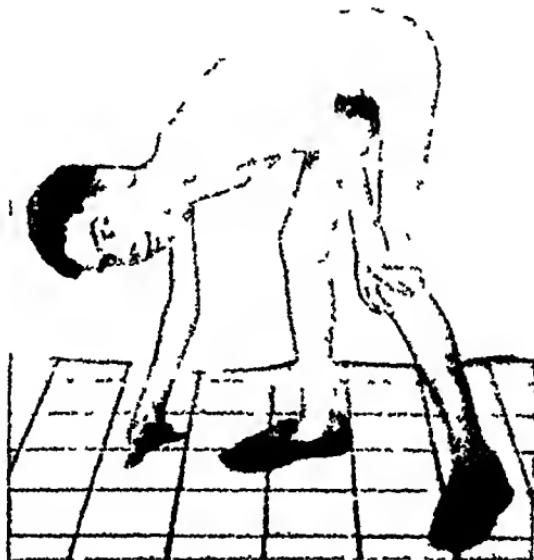


Fig. 262.—Pseudohypertrophic muscular dystrophy. A younger brother is similarly affected. Slight pseudohypertrophy of calves and characteristic method of assuming erect post on by "curling up oneself."

There are a number of diseases in which we have progressive muscular weakness, at times with atrophy, but of a character differing somewhat from that seen in the spinal form. Frequently in these atrophic muscles may be found infiltrated fat, giving rise to the appearance of hypertrophy, when the condition is known as pseudohypertrophy. These muscles in contradistinction to the ones affected by a lesion of the anterior horns, do

not show reaction of regeneration and such diseases are known as dystrophies, in contrast to spinal atrophies. They frequently are familial and usually hereditary. Of such lesions a progressive pseudohypertrophic dystrophy is a very common example (Fig 262).

Familial or hereditary diseases manifest themselves at times by changes in the peripheral mechanism, as in a dystrophy or atrophy and at other times produce changes in the central nervous system. For example, the afferent tracts in the cord leading upward to the cerebellum, such as occurs in Friedreich's ataxia, where we have ataxia, nystagmus and absent knee-jerks, pes cavus, extensor type of plantar reflex (Fig 263).



Fig 263.—The lower extremities of twins suffering with Friedreich's ataxia illustrating the pes cavus and dorsal flexion of toes.

If we have a patient who shows a spastic paralysis in the legs with increased deep reflexes and bilateral Babinski and in the upper extremities a flaccid paralysis with reaction of degeneration, atrophy in the muscles, and an absence of deep reflexes with no sensory changes, it is obvious that we must be dealing with a combined lesion of the lateral columns and the anterior horns. There is but one disease which may produce this namely, amyotrophic lateral sclerosis (Fig 264).

If we were to destroy the posterior columns subserving deep sensation, there would be produced an ataxia of sensory character due to the fact that the patient is unable to orient himself as to the position of his extremities and the extent and direction of their movements.

If we have a patient, therefore, who has a sensory ataxia, and, in addition, an absence of deep reflexes, with or without pain, and some loss of touch or pain sense, it is logical to assume that we have interrupted the deep reflex arc in its sensory portion, namely, at the posterior root, and, in addition, destroyed the posterior columns. Such a condition could be the result of only a tabes dorsalis or locomotor ataxia. The lesion of the posterior

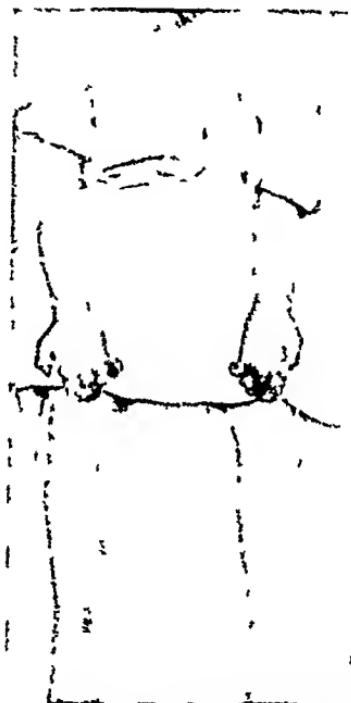


Fig. 264.—A case of amyotrophic lateral sclerosis, showing atrophy of small hand muscles

root would produce an absence of deep reflexes, spontaneous pain, and a loss of sensation to all modalities. Insomuch as deep sensibility is of more recent formation than pain, the long fibers in the posterior columns first undergo secondary degeneration, resulting in the loss of sensation of joint and muscle sense with ensuing ataxia (Fig. 265).

If, now, we are presented with a case in which we have a loss of joint and muscle sense, with a sensory ataxia and a weakness

with spasticity and increased reflexes, it is obvious that we are dealing with an involvement of more than one column of the spinal cord, and in such cases it would be a combined lesion of the posterior and lateral columns. Such a condition could be due to very few diseases, the chief one being combined degeneration of the spinal cord or posterior lateral sclerosis, which is almost invariably due to pernicious anemia. So if, in addition, to the neurologic phenomena, we discover a positive color-index and an achlorhydria, the diagnosis is positive.

Let us suppose a patient who has a paresis of his lower extremities, with increased deep reflexes, bilateral Babinski and



Fig 265.—A case of tabes dorsalis, illustrating the marked hypotonia resulting from a loss of proprioceptive (muscle, joint, bone) sense.

spasticity, in the upper extremities he has a flaccid paralysis with atrophy, reaction of degeneration, and absence of deep reflexes. In such a case it would be obvious that we are dealing with a lesion which affects the anterior gray matter at the level of the upper extremities and presses on the lateral columns.

If, now, we find that in the levels of the muscular supply of the upper extremities there is a loss of sensation to pain and temperature sense, but a preservation to touch, the diagnosis is apparent.

We will remember that the fibers subserving pain and temperature sense entering the spinal cord cross in the gray matter to the opposite spinothalamic tract. A loss of sensation to pain and

temperature sense and a preservation to touch, therefore, can be produced by a lesion about the central gray matter interrupting the crossing of these fibers, whereas the ascending fibers for touch remain unaffected in the posterior columns. Such a



Fig. 266.—A case of syringomyelia, showing characteristic scoliosis, atrophy of muscles of upper extremities, spastic paralysis in lower extremities, and a loss of sensation to heat, cold, and pain over the upper extremities and upper part of trunk.

condition may be due to but two diseases—an intramedullary tumor or syringomyelia (Fig. 266).

If we are confronted with a disease which produces a transverse lesion of the spinal cord it is obvious that at the level of the lesion, having interrupted the deep reflex arc, such a reflex would be absent. Having affected the anterior horns, that would

destroy trophic function for that particular segment of muscles, and here we would find a flaccid paralysis with reaction of degeneration and atrophy. This level is necessarily very narrow, and often these signs may not be elicited.

Interrupting the pyramidal tracts we would produce below the level of the lesion a spastic paralysis with increased deep reflexes and pathologic reflexes.

Having interrupted the ascending sensory pathways at the level of the lesion, we would have a loss of sensation below that level. The bladder musculature corresponding in a measure to the somatic musculature, would inhibit a sphincteric hypertension producing a retention of urine. Such a lesion could be produced by myelitis, tumor, thrombosis, hemorrhage, abscess or compression of the spinal cord, or by disease of the spine, such as Pott's disease.

The onset of such a condition frequently allows us to rule out a number of diseases. For example, the vascular diseases are sudden in onset. Tumors and compressions are slow in onset.

Again referring to some anatomic facts, we find considerable assistance in the differentiation of transverse lesions of the spinal cord. The fibers for pain and temperature sense, as they enter the spinal cord and cross, ascend in a lamellar manner, those forming the lowermost segments are gradually pushed outward by the increased number of fibers from upper segments. So when we deal with an intramedullary lesion in the cervical region, if it be incomplete, the outermost part of the spinothalamic tract is intact, and as this represents the lowermost segments we will find that the saddle area supplied by the sacral segments remains unaffected as to sensation.

On the other hand, if we are confronted with a lesion producing a compression from without, the lowermost segments are first affected, so that there is an analgesia up to, but not including, the last three or four segments below the level of the lesion.

In a transverse myelitis there is a complete loss of sensation below the level of the lesion. In the diseases on which a transverse lesion is produced by gradual compression there is a complete loss of sensation several segments below the level of the lesion,

and a diminished amount of loss of sensation up to the level of the lesion

Just as a small amount of knowledge concerning anatomy and physiology permits us to diagnose diseases of the spinal cord, so we find a corresponding assistance in diseases of the brain



Fig. 267.—A case of Weber's syndrome. Left-sided paralysis of the third nerve (ptosis) and a right hemiplegia resulting from a gummā at the crus cerebri.

stem and the rest of the central nervous system. We remember that the pyramidal tracts cross at the pyramidal decussation in the medulla. The motor fibers subserving the motor cranial nuclei cross at much higher levels so that if we have a lesion in the brain stem at the level of the seventh cranial nerve it will

produce a paralysis of the facial muscles on the side of the lesion, but inasmuch as the pyramidal tract decussates below this, there will be a hemiplegia of the opposite side of the body. These crossed paralyses always indicate a lesion of the brain stem and may involve all of the motor cranial nerves, such as the twelfth, seventh, sixth, third, and so on.

Many of these crossed paralyses bear the names of clinicians who first discovered them, but it is useless to burden yourselves with their names at this point. Suffice it to say, in pointing to this condition, that if we have an ipsilateral paralysis of the third nerve and a contralateral hemiplegia due to a lesion at the crus, where the third nerve crosses the pyramidal tract, the condition is known as Weber's syndrome (Fig. 267).

You may recall that the motor cortical representation is located in the precentral gyrus, or rolandic area, and occupies a wide-spread portion of the forebrain. If we are confronted with a case in which we have a wide-spread paralysis involving the face, arms, trunk and legs, with evidence of there being only a slight lesion, it would be illogic to seek for such a lesion in the motor cortex. It would be much more logical to search for it at some place where these fibers converge, and such a place is found in the internal capsule. Here the hemiplegia may be associated with a hemianesthesia, or a hemianopsia. If we find a case with a hemiplegia, hemianesthesia, and hemianopsia it is almost certain that we are dealing with a deep-seated lesion involving the internal capsule.

Certain so-called centers may be found in the cerebral cortex, centers for motion, sensation, certain parts of speech, vision, smell, etc., and a destruction or irritation of such a part produces symptoms which lead to the diagnosis.

Many diseases of the brain produce, among other symptoms, an epilepsy or a modification of consciousness, with or without convulsions. The aura, which is the first symptom of such an epilepsy, is dependent upon the part of the brain involved. There may be a motor or a sensory aura. A motor aura consists of certain clonic twitchings of an extremity or a part, or a cortical fit, Jacksonian epilepsy.

If, therefore, we have a case in which an individual develops a generalized convulsion preceded by clonic twitchings in the right side of the face, we may conclude that he is suffering from disease in the precentral gyrus in its lower part, affecting the face center.

If, on the other hand, a patient exhibiting such a convulsion presents as his aura a disagreeable or disgusting taste or smell, we are dealing with a lesion of the uncinate gyrus, and the convolution is known as an uncinate fit.

If, preceding the convulsion, we have an aura which consists of visual hallucinations, when they are crude, consisting of flashes of light, it is likely that we are dealing with an occipital lobe lesion. In such a case the hallucinations are frequently of the homonymous hemiopic character, and when examined the patient will be found to have a homonymous hemianopsia.

If, on the other hand, the aura consists of hallucinations which are integrated, such as moving men or animals, it is likely that we are dealing with a lesion of the temporal lobe.

The various aphasias obviously easily point to a localization of their particular pathology.

You may remember that the optic nerve proceeds backward to the optic radiations, crossing at the optic commissure, so that a lesion behind the commissure affecting one optic nerve produces a blindness of the corresponding halves of both eyes. On the other hand, a lesion in the central part of the optic commissure will produce a blindness in the temporal halves of both visual fields. Such a lesion usually is due to a pituitary tumor, or a suprasellar cyst, or a cystic third ventricle.

The central nervous system of man is the result of the superimposition of continuously newer developed parts. Beginning with a very low form of organism the simplest types of reflex movements existed and as newer reflexes were added they inhibited the older and simpler types.

For example, if we produce a lesion of the brain stem somewhere about the level of the red nucleus, there are released a large number of integrated movements, such as running, swimming, standing which are dependent on reflex arcs, the sensory

elements of which may be deep sensation, labyrinthine impulses, etc. Motion as willed by the motor cortex is associated with tone, which is influenced by or regulated by the older mechanisms found in the gray ganglia of the brain in the lenticular nucleus.

Lesions, therefore, of this area give rise to a number of motor disturbances, chiefly of the hyperkinetic type, which are the



Fig. 268.—A case of parkinsonian syndrome produced by encephalitis lethargica, showing the attitude, immobile facies, and rigidity.

released integrated movements of lower parts of the central nervous system. So we see choreiform movements and various dystonias producing myoclonic jerks as the result of striatal body disease and a certain increase of plastic tone, with or without tremor in diseases of the globus pallidus producing the syndrome which is recognized as Parkinson's disease (Fig. 268).

There remains but one more consideration. As a movement is willed, each muscle which is contracted is held in check by an antagonist, so that the movements are correctly timed, and are properly directed and extended. Under certain conditions that part of the central mechanism which is concerned with the contraction of antagonistic muscles, the cerebellum, is affected by disease, and there results an inability to smoothly perform motions.



Fig. 269.—A case of thrombosis of the postero-inferior cerebellar artery. Both sides of the face were sprinkled with talc, which adhered only to the sweating side. On the right side may be seen a narrow palpebral fissure, small pupil, and absence of sweat (Horner's syndrome). The right side of the face also showed a loss of all sensation. There was a cerebellar ataxia on the right side of the body and a diminution of pain and temperature sense on the left side.

This gives rise to an ataxia which differs from that of locomotor ataxia in that there is no loss of sensation. This ataxia is characterized by dyssynergia, dysmetria, and dystonia, and because of the adjacency of the vestibular nuclei which subserve the functions of the labyrinth the ataxia frequently is accompanied

by vertigo and nystagmus and abnormal positions of head and body

Because of the adjacency of such structures the diagnosis of small lesions in the brain stem is very frequently made possible. For example, if we are confronted with a case in which we have a sudden onset, speaking for a vascular lesion which produces an analgesia of the right side of the face, often with absence of sweating on that side, a right-sided cerebellar ataxia and a left-sided hemianalgesia, we are dealing with a lesion in the circumferential area of the pons, which is supplied by the postero-inferior cerebellar artery.

Such a condition usually is due to a thrombosis of this artery, which produces a destruction of the spinal-cerebello, spinothalamic tract, and the descending sensory root of the fifth nerve (Fig. 269).

In this very superficial and rapid survey I have attempted to point out that with a knowledge, not particularly extensive, of the important tracts, reflex levels and centers of the central nervous system, one is able by the grouping of a few signs and symptoms, to accurately localize a lesion. Once a lesion has been localized, the diagnosis is a simple matter, inasmuch as but few diseases produce pathology in certain localities of the central nervous system.

I would, therefore, recommend to you that the method of learning neurology consists not in the memorizing of pages of text-books, but in familiarizing oneself with certain anatomic and physiologic facts, and in the exercise of ordinary logic and horse sense.



CLINIC OF DRs SOLOMON STROUSE
AND PHIL A DALY

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DIABETES AND PREGNANCY

DIABETES mellitus as a complication of pregnancy is in our experience, comparatively infrequent. Sugar is found in the urine of pregnant women frequently enough, in the majority of cases the reducing substance is lactose. One group will show a small amount of urinary glucose, a normal blood-sugar, and with no real parallelism between secretion of sugar and intake of carbohydrate. This group has received considerable attention in the literature for such individuals possess the syndrome characteristic of the anomaly known as "renal glycosuria." The cases we are going to discuss today are true diabetics, and each one will be presented to stress certain definite points. At the present time the use of insulin certainly has made the combination of diabetes and pregnancy much less formidable, but, as we shall show, it has in no way diminished the need for strict individualization and careful dietary control. We still see mild types of diabetes in the pregnant woman, in fact, we see women apparently diabetic only during pregnancy, and on the other hand, we see pregnancy as an intervening complication on the burden of diabetes. It is perhaps true that the combination of diabetes and pregnancy demands stricter attention to detail than uncomplicated diabetes, proper orientation of the case is always an essential foundation for successful therapy.

CASE I

Mrs E, twenty-eight years of age, a primipara, three months pregnant. Has been feeling perfectly well until the past week,

when she has been passing large quantities of urine and drinking much water Her appetite has always been good and she has a decided fondness for sweets, there has been no increase in appetite recently For the past three or four days she has been dull and drowsy, but at the same time unable to sleep Otherwise her history is negative

When she entered the hospital she was quite disoriented in her surroundings, her breath had a marked fruity odor, the urine contained much glucose (36 per cent), and was heavily laden with acetone and diacetic acid Blood sugar 180 mgm per 100 c c

Physical examination negative except for the pregnancy

A diet of skimmed milk, fruit juices, and coffee for twenty-four hours caused a definite clinical improvement in the patient and a diminution in the amount of acetone and diacetic acid in the urine In forty-eight hours the acidosis had entirely disappeared, and on a diet of protein 50 gm, carbohydrate 50 gm, and fat 75 gm the glucose disappeared from the urine in a few days The patient left the hospital on a diet from which only sugar and heavy pastries were excluded

This patient could not be controlled, and throughout the remainder of her pregnancy, it is reported, she ate about as she pleased, but had no further trouble and delivered a normal healthy baby

This patient probably had a very mild diabetes which under ordinary circumstances gave no trouble, but which during her pregnancy suddenly flared up and under improper diet control developed into a condition of acidosis which could easily have become serious Relief was promptly obtained by simple regulation of diet, and though the patient was guided only by her own appetite, the remainder of her pregnancy was not again attended by a recurrence of acidosis, nor did any harm attend the fetus We have no way of knowing whether or not she was a real diabetic However, during the course of her pregnancy some insult (what we do not know) to her metabolic processes produced a temporary breakdown in the carbohydrate metabolism, with the resultant acidosis and threatened coma described above

CASE II

Our second case is that of Mrs B, twenty-eight years of age. This was her third pregnancy. Both previous babies died soon after birth, one lived forty minutes, the other, ten hours. During both former pregnancies patient had sugar in her urine from the early months and persisting throughout *in spite of rigid diet*. During the whole course she felt perfectly well until induction of first labor which was two weeks overdue. During this labor she had convulsions and albuminuria. In the interval between the second and most recent pregnancy one urinalysis showed no sugar.

When first seen she was four months pregnant, felt perfectly well, physical examination negative. There was a small amount of glucose in her urine. Chemical analysis of blood specimen revealed sugar content of 88 mgm, non-protein nitrogen 31 mgm and uric acid 3.3 mgm. Blood Wassermann was negative. Mosenthal test for renal function was normal.

A meal high in carbohydrates increased the blood-sugar to 138 mgm after one and a half hours, and glucose appeared in the urine. Continuation of a full diet produced from 1.6 to 5 gm of glucose in the urine. During the fifth and sixth months she was allowed a general diet, excluding only rich pastries and sugar, during that time the urine showed never more than a trace of glucose. In the seventh month she ate everything without restriction, the sugar content of the urine increased to 28 gm in twenty-four hours, and of the blood to 143 mgm, specimen being taken in midafternoon with no preliminary starvation. For the remainder of her pregnancy sweets were excluded from the diet, the sugar content of the urine never exceeded 0.6 per cent. On one occasion following a luncheon which included malted milk the blood-sugar rose to 157 mgm.

On account of a small pelvis delivery was by cesarean section, with a healthy baby as a result.

This case demonstrates a mild diabetes which had been rigidly controlled by strict dieting during two pregnancies, both of which had terminated unfavorably, both babies were lost. During this pregnancy the patient was treated as a very mild

diabetic, kept under careful observation dieted more leniently, even to disregarding an occasional trace of sugar. The outcome was a happy one.

During the non-pregnant state this woman showed no evidence of diabetes, but pregnancy throws enough extra strain upon the metabolic functions to produce a mild glycosuria. This requires a slight restriction in diet rather than a rigid limitation. It is, indeed, possible if not probable that the poor results obtained in the earlier pregnancies may have been due to too rigid dietary restrictions, which ended in disturbing the dietary balance.

CASE III

Mrs C is a patient thirty-two years of age, this is her fourth pregnancy, she has 2 living children and has had one dead fetus at term seven years previously. At that time she had sugar and albumin in the urine. Attention was first directed to the glycosuria three years ago, when itching of genitalia prompted an examination of the urine. There were no other symptoms of diabetes. At that time the sugar content of the blood was 300 mgm and the sugar in the urine seemed to vary greatly on the same diet. By maintaining a moderately strict diet she remained sugar free during the past year.

When first seen she was about four months pregnant physical examination negative, urine heavily laden with sugar, no acetone bodies, and no albumin. After two weeks of limitation of carbohydrates the sugar in the urine disappeared the blood-sugar determination revealed 151 mgm per 100 c.c. During the remainder of the course of pregnancy the glycosuria was controlled by dieting rather strictly, but without the use of insulin.

Near the time of her expected termination of pregnancy she suddenly developed an albuminuria and hypertension. Labor was induced and her pregnancy consummated successfully. When she left the hospital her diabetes was under control by dietary measures.

This patient represents a moderately severe diabetes of several years' standing. She became pregnant and the diabetes apparently became aggravated. However by using a carefully con-

trolled dietary scheme, she avoided any trouble which might have resulted from her diabetes. She completed her pregnancy unharmed.

CASE IV

Our last case is that of a rather mild diabetes which, neglected, developed into a serious state of acidosis and coma.

Mrs R., a woman thirty years of age, a primipara at term. She gave a history of glycosuria during the early months of her pregnancy, for which she dieted haphazardly for a short time, but for the most part was negligent both in diet and in seeking medical advice. The urine, examined at infrequent intervals, sometimes contained sugar, at other times did not. The most recent examination, some weeks previous to admission, contained no sugar, but did contain albumin.

She entered the hospital in a semicomatose condition, with very rapid pulse, pronounced Kussmaul breathing, very sweet fruity breath, diminished intra-ocular tension, and some vomiting. She was barely conscious, the only stimulus to which she would respond was thirst, and she would take fluids almost continuously. Heart tones of the fetus could not be obtained. The urine contained between 4 and 5 per cent of sugar and showed marked acetone and diacetic acid reactions. Blood-sugar estimation revealed 330 mgm per 100 c.c.

Treatment with insulin was begun at once. Being ignorant of her previous diet and having no blood-sugar determination immediately, fruit juices, to some of which sugar was added, and skimmed milk were freely given. Insulin in doses of 15 to 20 units was given every fifteen minutes, 70 units being given in the first hour, and 40 units in the second hour. At the end of this time there began to be an improvement in the mental condition of the patient, and the dosage was set at 15 units every three hours.

During the first two hours of insulin therapy every specimen of urine voided was examined. Thereafter a specimen was obtained and examined especially for acetone bodies one-half hour before each dose of insulin was given. The quantity of sugar increased, but after three hours, when 110 units of insulin had

been given, the acetone bodies were definitely diminished, and this diminution continued as the clinical condition improved

After six hours the patient had recovered from her comatose state, and the acetone and diacetic acid in the urine were much diminished. In twelve hours the acetone bodies had almost entirely disappeared and there was no clinical evidence of acidosis. The blood-sugar was higher than previously—430 mgm.

The patient went into labor and spontaneously delivered a macerated fetus.

Dieting plus insulin promptly eliminated sugar from the urine, the blood-sugar dropped to 190 mgm. On the day when the patient left the hospital, the blood contained 110 mgm per 100 c.c. This patient apparently had a real diabetes, the seriousness of which she had not realized, as a result she was neglected during her pregnancy. The grave symptoms occurred rather suddenly, whether before or after the death of the fetus we do not know. She rapidly developed an acidosis and drifted into coma, which event previous to the discovery of insulin would, in all probability, have terminated fatally for her as well as her child. The efficacy of insulin here was unquestionable, it saved the mother, but, of course, could not undo the damage that had already been done to the fetus.

These four cases illustrate very well the points made at the beginning of this clinic. Diabetes and pregnancy never can be grouped into a system for routine treatment, each patient requires careful diagnostic analysis and careful therapeutic planning. The presence of sugar in the urine of a pregnant woman should awaken the interest of the physician in diagnostic possibilities but should not send him into an uncharted path of drastic therapy. From a study of our own cases we believe that as much harm can result from injudicious drastic treatment in the mild cases as from neglecting any of the necessary measures in the severe cases. None of the lessons learned from handling the non-pregnant diabetic should be forgotten, and, on the other hand, we have to realize how frequently pregnancy upsets a normal metabolic balance. A woman, perhaps fundamentally so constituted as to carry her usual burdens, may, as a result of

pregnancy, show a definite diminution of carbohydrate tolerance. Such a patient will usually manage her pregnancy with a diet just below her tolerance, in other words, during pregnancy she becomes a mild diabetic and should be so treated. If, on the contrary, our pregnant patient is a diabetic with low tolerance, insulin may be required in order for her to receive enough calories to supply her needs. When insulin is used, it should be used with all the care, and all the dietary control which the experience of the most careful observers has shown to be necessary. The adoption of such general principles will make the problem considerably simpler than it seems to be at present, and will lighten the task of treating the pregnant diabetic.



CLINIC OF DR RALPH C HAMILL

WESLEY MEMORIAL HOSPITAL

DISABILITY, DAMAGES, OR DISEASE

THIS is an industrial as well as a medical problem. It is an inherent element in the situation of men working for other men. Ages ago men began to have to work for other men, at first as slaves or captives of war. Now the employer of labor looks benignly upon his force and expects the men thereof to acknowledge their indebtedness to him because he gives them work, and so he cannot understand why labor acts as it does. One of the places where flaws are certain to be found by anyone looking for them is in this phase of the relationship where real interest, such as comes only to men concerning something which is truly their own driving interest, would keep them at their work, but lack of it would allow the resentment of the injured to control the situation.

Examples of men suffering injury, but yet going on with what they are doing, are on all sides. They are so common that we cease to recognize them for what they are, namely, their own interest overcoming physical discomfort. What farmer hurting his hand in a threshing machine thereupon quits his job, claiming total or temporary disability? If asked why he does not lay off we would expect him to say "Why should I? I might as well be doing this as anything, and anyway, who would do it if I didn't?"

We medical men are asked to say when a man is disabled as the result of an industrial accident. We have two varieties of information to lead us in our judgment—what the man tells and what we see for ourselves. The former consists of the history, the latter of the examination. In the taking of the history

there are certain elements to which we must pay careful attention. They belong as truly to the examination as do the reactions of cranial nerves or tendon reflexes.

First of all is the manner in which the history is given. If there is any suspicion that the claim is to include a claim of mental disability, such disability often may be ruled out by first getting a more or less detailed account of the claimant's life, long prior to the accident. That is, men may give in a frank, convincing manner the details of school life, early working life, marriage, and so forth, and yet when they come to the discussion of the accident, claim failure of memory, inability to recall dates, names, and places. Such an inconsistency throws a shadow on the claim that such disability depends upon physical injury to the brain. This requires one qualification. In case of severe injury to the brain there may be a retrograde amnesia, followed by a period of unconsciousness, of failure of memory of the time both before and after the accident, but when a man has been injured and is not badly demented he can be depended upon to remember the details of the accident as they have been given him by others. By the time he comes to the consultant it is safe to conclude that he has been told all about the accident. So in cases of severe injury, in the absence of severe dementia, the rule is that the patient will himself tell of the retrograde amnesia, will comment upon it in such a way as in itself shows a perfectly good mental grasp of the situation. Such a grasp is, of course, inconsistent with the claim of failure of memory.

This sort of inconsistency is seen only in the very stupid, the laborer, for it is only the stupid who has complete faith in the value of a statement. That is, when a man insists upon being believed in a statement that is inconsistent with the rest of his story, it is because he fails to appreciate his doctor's ability to see behind the statement. Children have this belief in the value of the spoken word. The wisdom of the adult is largely dependent upon growing out of it.

Then, there is the general manner of telling of the events leading up to the accident, as well as the details of these events. For example, in the cases I am going to describe to you the

events leading up to the disability were recited with an expression of a sense of injustice and resentment that was unmistakable It is as true an expression of hurt feelings as the cry of the small boy who has been knocked down by another boy on the soft turf and so failed to get the ball both were running for This and the matter of damages are the most important elements in the picture It is the hurt feelings that demand satisfaction, and they express themselves in these statements of the circumstances attending the accident or the subsequent care by the company, its doctors, or the insurance company The man demands satisfaction This, of course, is a very different thing from physical disability

This resentment is to be seen in some cases taken from my records, in which I have tried to report to the doctor who referred the patient what seemed to me the essential details of the case

Case I—"In 1919, after a siege of double pneumonia, P B had his first attack of so-called chorea This attack lasted for about eight months He says that the attack was brought on by overactivity during convalescence 'The doctor told me that I could get up, but he thought I would set around, instead I went right to work *He should have told me not to go to work*' Eight months ago there was another attack of 'chorea' without any known cause One afternoon about 2 30 he was carrying a boy 'weighing about 100 pounds' He had to go down a flight of very narrow stairs slipped on the top stair and slid down, the boy on top of him At the bottom he felt chagrined and 'all in,' but picked himself up feeling as though there was a burn on his arm and a burning sensation on the back However, he carried the boy across the street under his right arm and went back to work He started to type, but it did not go very well He went home and had to lie that night on his stomach because of the burning sensation in his back The next day he saw a doctor who advised hot packs on the back He worked until about 11 o'clock and then went home to apply the packs He was back at work at 3 00 P M The next day, Saturday, he did

not work, but went to the store to help others with his work. On Monday he was sent to the hospital and put to bed. After three days in bed he began to feel very nervous and 'rigid'. That night he rang for the nurse and she was so busy she couldn't come. The nervousness got worse and worse, the nurse did not come and finally he threw a magazine on the floor to attract her attention. The spell lasted an hour or two, at the end of which time he cried. Since then, approximately a week, he has had from one to three attacks a day.

"Examination showed a man having constant twitchings and jerkings, grimacings, and rubbing of the hands. He was almost constantly in motion and much of the motion was with extreme tenseness of muscles. During the time when the movements were most marked there would be a considerable increase in the rate of heart beat. The facial expression during these times would be one of rather marked apprehension. He was in good flesh and had a good color. The reflexes, obtained with much difficulty because of the more or less constant motion, were normal. The cranial nerves were normal.

"The character of the movements stamps them conclusively as of functional and not organic origin. There was nothing that indicated disease or damage to the central nervous system. On the contrary, a few of the man's statements clearly indicated the essentially neurotic disposition of the individual. Such as, for example his criticism of the doctor for allowing him to go back to work, his criticism of the nurse for not coming when he rang for her, his effort to make me understand the nature of his accident in telling me that the box which he carried weighed 100 pounds, a thing not probable or consistent with the statement that he carried it under his right arm after the injury.

"It is my opinion that this man is a neurotic. He has had three attacks similar to the present one—one following pneumonia, one without cause, and the present one. It is possible that the attack occurs at this particular time because of the accident just as an accident may unnerves anyone. However, such an unnerving should not last as it has in this case, and would not if the patient were not a neurotic. In this sense the present

attack is caused by the accident and in this sense only I believe a settlement would help to clear up the condition, and yet I cannot in truth say that the present condition is due merely to desire for a financial settlement ”

Case II — “This morning I examined R D , colored He gave me the following account of his disability About six weeks ago while pushing a heavy weight his feet slipped out from under him, causing a hyperextension of the back He fell to the floor with a sensation of something breaking loose in the small of his back He went immediately to the doctor’s office, but says, ‘*He didn’t do nothing but give me some pills and a bottle of liniment and tell me it’d be all right in a few days*’ He then went home and to bed The next day the doctor came from the Company, but ‘*He didn’t do nothin’, didn’t even set down, just asked me how I felt*’ His own doctor came and gave him external and internal treatments He then stayed in bed a couple of days and then got up and sat around home, reporting to the doctors at the Company’s office every two days He is now in practically the same condition as when hurt ‘*The doctors at the office and the special doctors they sent me to down town said that I went too long, seven months without the proper treatment*’ When pinned down he said the masseur made this remark, and then he said, ‘There wasn’t anyone else to say it—my doctor said it

“Examination was of a large, obese negro The cranial nerves are normal station is normal As he walks he limps slightly on the right leg He can stand on either leg alone, though when he does so complains of pain over the lower right sacral region and just below the right gluteal crease However, as he walks there seems to be no resistance to the normal rather abrupt fall of the pelvis as it shifts from one leg to the other When asked to bend the back in any direction, he refuses to try, complaining that it causes too much pain, yet when he sat down to put on his shoes he bent his back, flexed and rotated his thighs, in a manner normal for so fat a man The ankle-jerks are diminished, the cremasteric reflexes are diminished, otherwise the reflexes showed nothing of note

"I give in detail certain factors in the history because I believe they clearly express a mental attitude on the part of this man which is, if not diagnostic, at least strongly suggestive of functional disability. By 'functional disability' I mean disability not dependent on organic causes. I cannot say that there is nothing abnormal in the right sacral region, and I cannot say what the relationship of the accident is to any existing abnormality, but from the manner of the patient it is obvious that he is laboring under the idea that he has been badly treated and deserves recompense therefor. This is an attitude of mind almost invariably encountered in this class of individual who is seeking damages. As to the actual disability, I could find no evidence of the presence of such a disability as would remain stationary over such a long period of time. There is certainly nothing in the central nervous system to indicate such disability. I am certain that the functional disability is being used with the hope of securing damages."

Case III—"I found K. E. in bed clothed only in a nightgown. She told the story of her disability, injury, and accident in the voice typical of the injured and complaining woman. I was unable to get a description of an accident that sounded as if it had been a serious one. About two and a half months ago she was struck by a hand truck pushed by a boy and squeezed between the truck and a post in the plant where she was working. It was impossible to get a definite statement of unconsciousness or immediate disability. In fact, according to her statement, she was taken by the foreman to a seat, left there a short time, then in a taxi to a doctor's office. At the time of the accident the chief complaint was of the right knee. Three days later, however, it was of pain around the right chest, which has increased 'more and more every day.'

"Since the accident she has been unable to work because of the pain in the right side.

"Examination showed nothing abnormal. There were no abnormal sounds in the chest, no signs of pleural effusion, and I was unable to find any local point of tenderness such as would

indicate a fractured rib. The complaint was rather of unbearable tenderness of the entire right side of the chest. The pain was declared to be worse whenever she attempted any movement. However, when asked to take a deep breath the right side of the chest moved equally with the left indicating an absence of injury to the physical structures of the chest wall. If exercise did cause pain there should have been a lagging and failure of movement of the right side of the chest in this deep breathing.

"It is my opinion that the fact that this woman was deserted by her husband plays a part in her condition. She was left without assistance and had to work to support her two boys—five and two. This accident has perhaps precipitated a depression and has been followed by a state of disability for which there is no physical basis. It is impossible to say what sort of a woman she was before the accident and how much she has changed since. She made but one allusion to damages. Her complaint was more of pain. Once she said that the pain made it impossible for her to work and she had to work to support her children, and that if she could not work where was the money to come from?"

Case IV—"Yesterday I examined J J at my office. He gave a comprehensive, detailed statement of his accident, showing complete retention of memory of the accident. The account was colored by the fact that he wished me to understand or perhaps better, was persuaded himself, that injustice had been done him. For example, he stated that the foreman was doing work on a boring machine *that should have been done on a lathe*, was working with a *file instead of a chisel*, and the wheel he was working on in some way slipped and struck J on the forehead. He stated that when he woke from a period of unconsciousness of about two hours that he was lying in the doctor's room, and upon waking cried. As he made this statement his eyes filled with tears. The day following the accident he had developed what he described as two big balls behind his right ear and one behind the left, he had a headache at the back of the head and through the temples, and said he was *told by the insurance doctor that all this amounted to nothing*. He was then sent to a doctor on LaSalle Street—

'That wasn't no doctor, he just held my head and another walked around me and asked me questions and said that in six or seven weeks I'd be all right'

"Since the accident if he walks fast or thinks hard it hurts him through the temples and through the back of the head, and he also has pain in the heart which prevents his sleeping. Physical examination showed a very well-built, well-preserved man in the early thirties, with no sign of injury other than a small scar about an inch long above the bridge of the nose. However, his pulse-rate, taken twice during the examination, was 120 per minute. Blood-pressure, deep and superficial reflexes and the cranial nerves, including the optic nerves, were normal. When I was examining the pupillary reflexes I asked him to look at the top of the window, which demanded bending his head back. He thereupon cringed and intimated that I had acted without consideration of his feelings. When I attempted to explain the purpose of the examination and the fact that the reflexes and nerves were normal, he became excited, said that I was no doctor, and that I didn't know what I was talking about.

"Throughout the period of examination I was impressed with this man's vindictiveness toward the circumstances leading up to the accident and toward the insurance company. Later this was shown also toward me. The only abnormal finding was the rapid pulse. This could be produced by the apprehensiveness occasioned by the examination, and I can in no way see being due to the accident. It impressed me as a very unstable individual who, as a child would, was attempting to prove his case by assertion and the exhibition of emotion. Objective proofs of disability were completely lacking."

The second factor in the history has to do with the seriousness of the injury. Obviously, if the injury is slight, serious disability in the absence of complicating infection or what not, is not to be expected. If the injury is described as severe the severity is a matter to be judged by the examiner, like anything else in the examination. For example, many patients while describing the accident, let us say a fall on the head, while dilating upon the severity will, if directly questioned, speak of unconsciousness

as lasting anywhere up to two weeks but if the questioning is directed toward finding out their first memories following the accident such as how they got to the doctor, what he did what first happened in the hospital, and so on definite statements may be made with a naïvety lacking all the appreciation of the fact that knowledge of such details rules out subsequent claims of unconsciousness

For example the next case I will describe is of interest as indicating the prejudiced statement of the injured in which he attempts to demonstrate to the examiner the severity of his injury

Case V—"C B was born of American parents, but has never been better than a laborer, and has wandered from one job to another, after the manner of the inefficient. At one time he attempted to farm, but was unable to make a living even in that capacity. His home was as untidy as any home I have ever been in. It showed, in my opinion, a lack of mentality both on his part and that of his wife. There are 10 children, several of whom are old enough to have made it possible to have a decent home if the parental guidance had been there.

"B is fifty-one years old and denies any associated or collateral evidence of inherited disease. He quit school at twelve in the fourth grade, and has never made any progress up the economic ladder. He had been working for about ten months for the same company when he was injured. This, he says, was between May 15th and 18th, between 8 and 9 A.M., when, as he was picking up a piece of wood, he was struck by a piece of wood about 12 inches long 12 wide, and 3 thick, flung about 4 feet landing over the right eye and temple. He says that he was knocked unconscious and came to with someone grabbing his arm saying '*You are hurt awful bad, hurry up and go to a doctor*'. He walked up the track and street about four blocks alone met a man coming out of a garage who directed him to a physician's office. The woman in charge *exclaimed as to how badly he was hurt told the doctor over the telephone how badly the patient was hurt* and then finally sent him to another doctor, who also

said that he was *hurt pretty badly and that he had better take something for it*. This the patient would not do because he feared that the doctor might take his eye out. However, the doctor put a couple of stitches into the wound over the right eyebrow, whereupon the patient left, saying that he was going home. The doctor said, 'No, you go back to the shop, keep in the shade and keep out of the sun, and then the people there will know how you were hurt, and keep out of sight of outsiders.' In answer to my question as to why he said this, B replied to the effect that he did not know why it was said. I might here state that it was obvious from B's manner that he wished to impress me with the idea that the doctor and his employer would be interested in hiding the extent of his injury. There was no particular complaint except that while at the doctor's his head felt big, and then, when he wished to turn his head, it would cause an unpleasant sensation or pain to run up the back of his neck. In spite of these symptoms he worked daily until July 11th. Upon this date he wakened his wife at 5 A.M., shaking the bed, moaning, as she says, rubbing his feet together, stiffened out, and jerking all over. He came to in about half an hour, acted dazed, and after taking some medicine which the doctor had given he slept for twenty-four hours. Since this first spell there have been numerous spells, gradually becoming more frequent. During the past winter, when at work, he began to have a pain running from the head and neck into the shoulders. He gives a graphic description of a sensation as of a coal at the site of injury in the forehead, with pain running from it down into the shoulders and neck. A few days ago he had eleven teeth pulled.

"The present complaint is of three or four spells a week if not controlled with luminal, no strength, vertigo, pain in the arms, shoulders and neck, stiffness of the hands, an inability to raise his arms as high as his shoulders, and inability to close his fists.

"Examination shows normal cranial nerves, except that he did not confess to hearing the ticking of a watch, although he heard everything said to him even in a very subdued tone. The deep and superficial reflexes were normal. The optic nerves and

visual fields were normal He walked without difficulty He resisted any efforts to raise the arms to the level of the shoulders, to close his fists, or to sharply turn the head He complained some of tenderness upon pressure over the cervical vertebrae, the shoulder-joints and the small joints of the hands When sensation was tested at first it was difficult to say anything definite about it, but as the examination proceeded he gradually claimed a loss or diminution of sensation on the right side of the body, which loss finally became complete over an area of the right forehead and upper cheek, in the neighborhood of the injury This loss of sensation, however, was not genuine, because at times when I told him to say 'yes' when he felt the pin or the touch, and 'no' when he did not, he would promptly answer 'no' to each touch over the supposed analgesic areas

"From the description of the attacks I believe that this man has epilepsy However, that the epilepsy is due to the injury I do not believe In the first place, the force of the trauma seems to me entirely inadequate to cause skull fracture or damage to underlying brain tissue I cannot for a moment believe that he could have had severe cerebral shock and walk to the doctor's and conduct himself immediately after the injury in the manner which he recites Present examination gives no indication of right-sided brain injury There is no suggestion in the history of a jacksonian character in the fits No fit has been followed by a unilateral paralysis, as would be expected if they were due to focal injury

"On the other hand, the attitude of both the man and his wife seem to me the opposite of naive When I asked him to take his shoes off his wife interfered with his attempts, telling me that he could not undress himself, and proceeded to do it for him As he assisted her he moved his hands much more than he did for me when I was examining the motion of the hands directly The sensory loss is not anatomic I can consider it only as evidence of the desire to impress the examiner with the extent of the injury and the severity of the disability

"In my opinion B is a man of inferior mentality who is having epilepsy as a result of beginning arteriosclerosis The

difficulty with the shoulders is certainly not as great as he tried to make me believe, but it was bilateral and was not confined entirely to the shoulders, being present also in the hands. I am much more inclined to believe that it is rheumatic rather than that it is due to any cerebral condition. I cannot see that any of this disability is due to the injury, which occurred almost two months before he ceased work."

To return to the matter of unconsciousness, there is a very decided difference between the physician's idea of unconsciousness and that present in the mind of the lay public. To the physician unconsciousness means coma. To the lay public it may mean anything, from coma to hysterical amnesia, so it is necessary when the patient says he was "unconscious" for a long period to have some corroboratory or opposing evidence, such as one patient saying "My wife said I was carrying on terrible and didn't know her." Another said, "The nurse told me they had to have help to hold me in bed." This obviously rules out coma and may be a manifestation of a personality defect rather than a sign of the severity of brain trauma.

Inconsistencies in the anamnesis may be obvious in any case where hurt feelings and a desire for recompense are present. This is as true of the man who really has something the matter with him as of him who has not. I have taken 60 cases from my records and divided them into three classes. First, those in whom I could find no evidence of abnormality. Second, those in whom functional abnormality was present and of a nature that seemed to me to rule out a direct dependence upon the complained of accident. Third, those in whom abnormalities of a physical nature are present, but obviously are not due to the trauma. There are 32 cases of the first class, those in which the complaints are purely subjective, 10 in the second class those in which evidences of emotional instability are present and 18 in the third, those in which signs or symptoms of disease are present, which, in my opinion, are not to be ascribed to trauma.

In the purely subjective group, the cases in which in spite of careful examination I failed to find any evidence of physical

disability that could in any way be attributed to trauma in most of these cases I found the resentful animus toward the circumstances that contributed to, or brought about the disability complained of In those cases I also believed I could demonstrate circumstances that satisfied me that the patient was magnifying or faking symptoms for the purpose of leading the examiner to form an erroneous and exaggerated opinion of the seriousness of the disability All three of these factors may be present in any one case, or only one may be made obvious in the hour that is usually sufficient to arrive at a definite opinion as to the nature and extent of the disability

Case VI—"E A , a teamster, was thrown from his wagon and injured by a street car He at present is suffering from convulsive attacks which I have demonstrated to my own and his doctor's satisfaction are functional I have been able to produce a convulsion by suggestion and have been able by means of physical punishment to stop the development of a convulsion that had gone about one-third the way There are no organic manifestations of a lesion of the central nervous system The patient's attitude is typical of that seen in the traumatic neurosis case, namely, a protestation that he is unable to control the disease phenomena (this on his own volition without question of mine), an attitude of a desire for satisfaction because of the suffering he has had to endure consequent to the accident

"It is my opinion that these attacks are purely functional, that the case is one of traumatic neurosis, and that there are no organic changes in the central nervous system due to the accident

"I am at present trying to bring about a cure There is some improvement, but I fear he will not give up his disease until definite settlement is accomplished "

Case VII—"L D , in company with 3 other men, was carrying a 450-pound plate of steel when the other 3 men all let go the plate and the entire strain came suddenly on his right arm He felt as though something 'busted loose' in the body of

the biceps, the elbow was jerked, swelled, and got stiff. He was away from his work for seven days. 'They put me back to work on the eighth day and about six weeks later I lost the use of the arm altogether.' The imputation that the loss was due to being put back to work was unmistakable from the tone of voice. 'I was told when they put me back to work that they would give me light work, but they gave me the same heavy work that I had been doing. My arm never was well—the doctors out there told me that they couldn't do my arm no good, the fix it was in, I'd have to get my own doctor. He gave me electric treatments, which do me good if I take three of them a week. One day I was taking thin sheets of steel. My arm began to get weak, I reached down to pick up a piece of steel, and it went dead all of a sudden. I went to the Company doctor. He examined it, gave me some liniment, and told me to come back the next day. Then he massaged it with some liniment, *that's all he done*. Then he'd have me come out there every other day and he would have five or six different doctors there to examine it.' After being sent to another specialist he returned to the Company, and '*They wanted to make a settlement with me. They wanted to pay me for three weeks and a half in full settlement, and I didn't accept it, and I haven't been back since'*'

"At present D complains that he gets no use of his right arm, that it is numb up to the elbow and that he can do no work.

"Examination shows no signs of atrophy. The measurements of the right and left arms 10 cm above and below the olecranon process are equal. He holds the right arm slightly flexed at the elbow and made no use of this arm or hand while in my office. However, he is able to make practically all movements with the fingers and arm, but they are of small amplitude, very feeble, and very slowly attempted. Once he used normal strength in attempting to approximate the elbow to the chest against my resistance, but on the second trial he made no exertion at all—at one time he showed normal power, at another no power. Passive movements are normal. There is a complete analgesia of the right hand and forearm, the upper level typical of the glove-like analgesia of functional disease. During the testing

with pinpoint I used a great many repeated jabs I then led him to believe that after testing him with electricity I was going to repeat the same performance with the pinpoint, but suggested that after the use of the electricity he would be able to feel the pin normally He followed the suggestion in a manner that showed his desire to escape further punishment From these reactions I believe that the declaration of analgesia was false

"It is my opinion that there is no organic disease of the central nervous system From the manner in which D told of his accident, the emphasis that he laid on the little done for him by the Company's physician, the fact that they put him back to work on the eighth day, and, more especially, his feeling as he told of the attempt of the Company to settle, I am inclined to believe that the continuance of the disability is entirely a matter of attempting to secure damages "

The 10 cases in the second class show signs and symptoms that are commonly met among the neurotics who have had no industrial accident Such symptoms as sweating palms and axillae, tachycardia, slightly increased blood-pressure, and so forth, indicating a functional abnormality that is to be considered on a basis of apprehension rather than on a basis of physical injury to the individual The apprehension may be a frank fear, as it is in the ordinary neurotic, a fear that can be best expressed as a fear of failure For example, if a man or a woman has been having a hard time carrying on their job, has been afraid they could not "make a go of it," either because the conditions of labor seemed too hard, because of advanced age or because of other factors in their life making living already a difficulty, such an individual who is already apprehensive may suffer an injury and such injury may be seized upon as a legitimate excuse for giving up what seems to be too hard a struggle There is also an element of apprehension about being examined The blood-pressure at the beginning of the examination may be higher than at the end This represents an emotional instability in the individual that deserves to be taken into account if one is trying to understand this problem of the industrial accident

spinal syphilis, 3 from the residue from disease in infancy, 2 are arteriosclerotics, 1 has the cord changes of anemia, 1 has a toxic neuritis 1 an arthritis, 1 intestinal worms, 1 a hyperchlorhydria of some sort into which I was not allowed to go, and 1 has an inguinal hernia. It is of interest to look at the subjective symptoms of the cases of syphilis with a knowledge of the symptomatology of cerebrospinal syphilis in mind. The 4 cases I will describe to you bring out the point I wish to make. For instance, in the first case an electric shock is followed by complaint of cramps. There is no question that electricity produces cramps, hence, the causal relationship is of interest. In the second case a mule stepped on a man's foot and caused gangrene to develop. Such gangrene of the extremity is seen frequently in cases of tabes in which there have been local injuries. The third patient lays the weakness of his legs to the effects of an explosion, and the fourth blames a slight knock on the head for a headache and subsequent blurred vision that came on a few hours later.

Case IX.—"I have examined R. B., who told me of having an electric shock while at work. Since this experience he has felt partially paralyzed in the left arm and leg and across the muscles of his left chest, claims he has had cramps in the left side, that he has had abscesses in his nose and head, that his left ankle is stiff and that he has almost continuous headache."

"Examination showed that the right pupil was practically normal, but that the left was double the size of the right, irregular in shape, and did not respond to light, although it did respond to convergence. There seemed to be some weakness of the right side of the face, but this was slight. The deep reflexes were active. Sensation was normal. There was a systolic murmur over the cardiac region, most marked just to the left of the lower part of the sternum, namely, in the region of the pulmonic valve.

"In view of these I inquired as to when he had had a chancre, and was told that he acquired his infection twelve to fourteen years ago.

"It is my opinion that this man is suffering from the late effects of syphilis I cannot see any evidence that would lead me to suspect injury to the central nervous system from any other source It is to be noted that there is practically no scarring of the left hand, with which he was supposed to have grasped the live wire I would insist upon an examination of the blood and spinal fluid, and, even though the reactions are negative, upon active antisyphilitic treatment "

Case X—"This afternoon I examined C B , who gives the following account of an accident that occurred about two years ago, when a mule stepped on his foot, mashing his left great toe This was followed by a gangrene of the toe and foot One month after the injury during which time he had been trying to continue with his work, the toe was amputated The end of the first metatarsal and the overlying parts have been sore ever since

"It is indicative of his mental attitude that when I asked him how he managed to keep up his work while the foot was sore, his answer was, 'I was on compensation pay They allowed me \$326 30, but the company and the insurance company kept back some and I only got \$180 30 ' I then remarked, 'You managed to keep at work pretty well,' to which he answered, 'Yes, but I suffered like everything I should have quit work before it got so bad Yes, it got very bad '

"For the past year it has been difficult for him to hold his water, at times some escaping He has suffered at intervals from sharp pains up and down the left leg He denies venereal infection, has been married for twenty years, and has 6 children

"Examination shows the left pupil larger than the right, both are fixed to light, but react to accommodation, Argyll-Robertson pupils The left lid droops slightly There is a slight Romberg sign The ankle- and knee-jerks are lost There is a loss of deep pain sense of the Achilles tendons, the testicles, and the ulnar nerves There is a suggestion of diminished pain sense around the lower chest

"It is my opinion that B is suffering from tabes dorsalis, invariably the result of syphilis I have had blood taken for a

Wassermann examination. The loss of the left great toe may be due to the trauma, or might be the result of a perforating ulcer. I do not feel that at present I can possibly determine that point."

Case XI.—"A few days ago I examined J. E., who stated that he had been entirely well until about two and a half months ago, at which time there was an explosion in a slag pit, and he was thrown 25 feet, his head cut in three places, but he managed to walk out of the mill and was at no time unconscious. In the hospital his wounds were dressed, he was in bed for a week, and then when he tried to rise he found that his legs were weak. He had had no difficulty with the sphincters, no particular headache and no pain.

"Examination showed a marked ataxia in the legs and a strongly positive Romberg sign. The left pupil was slightly irregular and did not react well to light, the right pupil, though not strictly normal, was better than the left. Ankle- and knee-jerks were absent, as were the deep reflexes in the arms. The right plantar was questionable, the left normal. The cremasteric and abdominal reflexes were normal. The optic nerves showed a stippling of the physiologic cup and a gray border. There was loss of pain sense in a zone 3 inches wide around the left chest, 1 inch around the right; there was also loss of pain sense over both lower legs. Heavy pressure on the Achilles tendons, testes, and ulnar nerves was well borne.

"Because of these findings I was convinced that this man was suffering from nervous syphilis and requested his being sent into the hospital, where, a week later, I did a spinal puncture and found 30 cells to the cubic millimeter, a positive Nonne reaction and both the hospital and another laboratory found a well-marked positive Wassermann reaction. The laboratory reported the Lange reaction of the tubercle type.

"Because of the above mentioned findings, which remained present at the time of the spinal puncture and because of the findings in the spinal fluid it is obvious that E. is suffering from cerebrospinal syphilis of the tubercle type. He has a long pig-

mented scar on the forehead, and one or two smaller scars over the occiput but I cannot see that these scars or the accident which occasioned them have anything to do with disability arising from syphilitic or tabetic changes in the spinal cord . Inasmuch as he has had no active antisyphilitic treatment, I feel that this should be instituted at once and that he would probably be better off for a rest of a week or two in bed ”

Case XII —“A few days ago I examined Mr K , who gave me the following history of his illness A few weeks ago at 4 15 P M , while working for an electric company, he stooped over and knocked his forehead on a wooden horse He was slightly dazed, but continued working until quitting time, washed up, and had no trouble on the street-car which took him several miles to his home He had supper and then left home on an errand When three blocks from home things suddenly went dark before his eyes and since then his vision has been blurred At first he had a slight, dull headache but this lasted only a few days There were no other subjective complaints

“Examination showed a paralysis of the right external rectus, the eye turned in In all other respects the examination was negative At my request this man was sent into the hospital, where I did a lumbar puncture The spinal fluid contained 15 cells per cubic millimeter and was positive to the Wassermann test This reaction and the number of cells confirm the diagnosis of syphilis of the nervous system Before the patient left the hospital I started him on mercury and potassium iodid

“It is my opinion that the paralysis of the eye muscle is due to syphilis and was not produced by the accident In fact, I believe that the accident had nothing to do with the appearance of the paralysis, and that the paralysis came on when it did in the regular course of the disease It was impossible to be sure of the origin of the paralysis without the lumbar puncture ”

The following case seems to be one of syphilis, but the accident may be the responsible factor

Case XIII—"Yesterday I examined Mrs L, who gave me the following details of her history. She is thirty-five years old and married first at twenty-two, her husband dying after five years. She worked in a shoe factory for five years, and married again three years ago, but has been clerking in a store where she was hurt.

"About ten months ago a man came into the store asking for a bar of soap. This was at 8:30 A.M. and she had just opened the store. The man went to the proper counter and got the soap and brought it to her. She wrapped it up for him, but they had an argument as to the price of the soap. She turned to the cash register, when he struck her with something on the forehead. She was knocked unconscious and remained so for twenty or twenty-five minutes. She then crawled out to the street and called for help. She lost much blood, was taken by the police to the nearest hospital, where she remembers the doctor sewing up her head. She was in the hospital two weeks. A week after her return home she began having dizzy spells, in which her head seemed to be swimming around, and the pain ran from behind the right eye to the back of the head. The first spell lasted about half an hour, and since then she has had these spells once or twice a week lasting from half an hour to a couple of days. In the past month she has had five of these attacks. Any stooping is apt to make her dizzy. She is afraid to do any work for fear exertion might bring on another attack.

"A few weeks after the injury she returned to her mother in another city and was sent to a doctor, who operated on her forehead, where he said he found a blood-clot. She was in the hospital only five days.

"Examination shows normal cranial nerves, normal station and gait and normal deep and superficial reflexes. There is a loss of sensation in the forehead above a linear scar which runs from one side of the forehead to the other (the operation was performed while she was at her mother's). There is a scar just above the hair line above the left eye. There is also a scar behind the left ear. There is a maculopapular eruption symmetrically distributed over both forearms. The blood-pressure is 100 systolic, 70 diastolic.

"This patient tells a perfectly consistent story of an injury to the head. Nevertheless, it seemed reasonable to have the blood examined because of the low blood-pressure and the skin eruption. The laboratory reported that examination of the blood revealed syphilis.

"Such dizzy spells as Mrs L. complains of can be the result either of the injury or of syphilis. Also, if they are due to syphilis, the injury may have caused the syphilitic infection to become localized in the brain. It would be necessary to submit the patient to thorough antisyphilitic treatment before one could say that the dizzy spells were not due to syphilis."

The last case I will describe is obviously one of cerebrospinal syphilis with no connection whatever between the injury complained of and the appearance of the symptoms. Why a man should expect damages under those circumstances is beyond comprehension, but perhaps is of interest as showing the lengths to which men will go in their attempts to secure recompense.

Case XIV.—"This morning I examined F. K. at his home. He says that he has felt badly for about a month. About five weeks ago he knocked his shin against a box in the dark. This produced a swelling a few inches below the right knee and I believe the skin was broken. He continued to work, however, and was treated by his wife at home. About three weeks after this injury he had an indefinite sense of pain in the left leg, but continued to work off and on for another week or ten days. One morning he tried to go to work, but after going a short distance he felt extremely dizzy, staggered, and had to return home. Since that time he has had continuous headaches, dizziness and weakness, and has vomited off and on. He has had no stools for six days, and this morning, when he tried to drink, water came out through his nose.

'Examination shows paralysis of the right fifth, sixth, and seventh nerves. It was impossible to determine, but there seemed to be some difficulty with the eighth also. There was nystagmus looking to the right with the left eye. The tongue protrudes to the right. There is an extreme ataxia of the left

arm and leg, a slight ataxia of the right. The deep reflexes are exaggerated in the legs, with a well-marked clonus on the left side. The right plantar reflex is normal, the left is abolished. The right abdominal reflex is normal, the left diminished. When he attempts deep inspiration there is a noticeable lagging of the left chest. The optic nerves are practically normal, at least there is no choked disk. There is a loss of pain sense on the left leg and lower trunk, less marked in the left arm and chest. The sense of passive movement is badly affected in the left foot, less so in the right.

"I cannot see that the present condition is in any way related to the slight injury to the left shin. There is a small abrasion at the site of the injury, but otherwise it seems to have healed normally."

"It is my opinion that this man is suffering from disease of the brain stem, which, in all probability, is of the nature of a new growth or tumor. From the rapidity of the development of the symptoms I am forced to suspect syphilis as the causative factor. In order to clear this point up it would be necessary to have proper laboratory examinations. There are other forms of tumor growths that could give this clinical picture, but until syphilis is eliminated K. should be given antisyphilitic treatment. It is impossible for his wife to give him adequate care, and it is absolutely essential that he be taken to a hospital. With the best of care, however, I would not be very sanguine of the outcome."

The symptomatology of these individuals who have been more or less severely injured, suffered therefrom for a period of time, and had to prove to a board of judgment that they were injured and disabled, is naturally largely subjective. For one thing, a period of time sufficient to allow of more or less complete recovery from such things as bruises, strains, cuts, shock, and so forth, has usually elapsed since the accident. With the lapse of time and the disappearance of actual disability, but with the need of proving such disability to a board of judgment the individual is driven to some kind of subterfuge, or to claim some pain,

numbness lack of sleep, weakness, and so on, or else acknowledge his ability to go to work. This alternative is abhorrent because of the resentment felt and unrequited, and because, at least in some instances of a fear lest, having experienced an accident which might cause permanent disability, they are returning to a situation in which they will be exposed to similar accidents. There doubtless are other factors that aid in determining the symptomatology of these unfortunates.

In going over my records I found that the symptom most commonly complained of is pain. This pain is usually in the neighborhood of the part injured. Where such injury has not caused or resulted in deformity, pain should have disappeared with the clearing up of bruises and abrasions. In other words, in two or three weeks at the outside. If one compares some of the harder bruises incurred in the college sport of football with those of the factory, one cannot help thinking that if the factory work was entered into in the same spirit as a football game the bruises would be taken in an entirely different manner. Pain in the head is the symptom commonly complained of following head injuries, that and dizziness. Of course, the complainant or claimant wishes to have it believed that the pain in the head is due to some kind of injury to the brain. In the majority of cases where such pain is complained of there is also a claim of loss of memory, lack of concentration, or other mental failure, this by way of backing up the idea that it is the brain that is injured. I do not believe that one pain in a hundred in the head has anything to do with the inside of the skull.

The complaint of dizziness is an extremely difficult one to meet. There is no way to prove it or to satisfactorily test for it. A person can claim dizziness and that is all there is to it. However, if one watches such a claimant during the examination and sees him change position in dressing, stooping to put on shoes, and then rising without any suggestion of discomfort, one has to discount such a claim. Partial paralysis of the arm or leg is another common symptom, apparently believed in by the patient or at least offered by the patient as an evidence of disability, in the belief that some injury to the central nervous

system or the nerves can account for such disability. Naturally, their information does not lead them to realize what tests there are to prove such nervous disability. The reflexes are usually normal or are increased on both sides of the body. There is absence of typical spasticity. There is failure in typical changes in gait, and so on, but perhaps the commonest pitfall of the claimant for damages is his lack of knowledge of the physiology of sensation. It is the easiest thing in the examination, as a rule, to demonstrate an anesthesia or analgesia of the legs or the half of the body complained of. If it is apparent that the case is one of plain seeking of damages, it does not seem to me justifiable to go into a thorough sensory examination because inevitably it leaves in the patient's mind one more thing to complain of. However, when it is necessary to test the validity of the claim and the honesty of the claimant, it is permissible to make a careful sensory examination. As a preliminary to such an examination it is easy to suggest to the patient what you, as the examining physician, would expect to find. That is, just before the sensory examination, while still examining reflexes or motor power or what not, the examiner can remark as much to himself as to the patient, "It's the left side that is affected," or the left arm or the left leg, and then when one starts the sensory examination it is possible to vary in a slight way the inflection of the voice, from which the patient implies that whereas he is expected to feel normally on the right side the left side should be numb. Surprise may be expressed at wincing or other evidence of feeling in an area where pain has been complained of, and so lead to a declaration of anesthesia or analgesia. At times it may seem best to first go hurriedly over the surface of the body without asking for responses and observe wincing as it occurs. Of course, one need see such wincing in the majority of cases only on the two sides of the body, and not be more exact than that. That is, if hemiplegia is associated with sensory change, such sensory change would involve one-half of the body. When the area complained of is less than one-half—an arm or a leg—wincing from a pinprick of the extremity is fair evidence of preserved sensation throughout. Of course, this is not strictly true, but

when such wincing is observed in such a hurried preliminary examination and later the point of the pin is borne without complaint and described as dull, such inconsistency invalidates the claim of disability at once. In fact, it is the inconsistencies in the sensory examination that seem to me most frequently to show the lack of honesty in the claimant. At least one may say and this seems to me of prime importance, that such inconsistencies demonstrate beyond the shadow of a doubt the effort on the part of the claimant to impress the examiner with an exaggerated idea of the severity of the disability. The symptoms include everything, from convulsive attacks, real and faked, melancholia or plain sadness, to poor vision, deafness, nervousness, impotence, and the signs of hyperchlorhydria.

DIAGNOSIS

It is difficult to speak of the diagnosis in these cases after the manner in which diagnosis is usually handled. As the two last classes of cases I have cited indicate, the injured individual may have symptoms quite irrespective of trauma, which symptoms, nevertheless, he tries to convince the world are due to trauma. The diagnosis in this instance is similar to that in general medicine namely to what noxious element must one ascribe the disease picture, but the chief function of diagnosis seems to me to lie in the discovery of the two factors already emphasized—the resentment against the company, employer, doctor, or insurance company that is expressed directly or indirectly, and the signs that one is justified in considering as pointing to the effort of the patient to impress the examiner with a false sense of the disability.

Cases I, II, and III show this in different ways. In Case I the patient stated and believed that the first attack was brought on by his overzealous activity during convalescence. "He should have told me not to go to work." It was the fault of the doctor that he had his first nervous symptoms. Also, the nurse delayed answering his summons, and the way in which he told of this expressed the suffering this delay caused him. The movements that were the outstanding symptoms and were complained

analgesia was given up when he realized how much suffering it was going to cause him.

In Case VIII, I put harder and rougher pressure on the patient's scalp when I was apparently examining her eye reactions than I did when examining the sensitivity of the scalp directly, but it was only during the direct testing that she complained. That there should be any degree of tenderness six months after such an accident also seems highly improbable. The mentally inferior type of the patient combined with the general attitude of the family convinced the girl that she had been very badly injured and must be recompensed for her suffering.

In connection with Case IX I would say, though I may be in error, that I have little faith in claims that disability of a chronic sort is caused by electricity. I think the shock either kills or the patient quite recovers.

Cases X, XI, and XII are frank cases of cerebrospinal syphilis, and the complaints are such as are expected in this condition. On the other hand, there clearly is no causal relation between having one's toe stepped on, even by a mule, and losing one's water for a year. To be sure, much might happen to a man who is thrown 20 feet by an explosion that would cause him to lose the strength of his legs, but when that loss is associated with loss of deep reflexes throughout and with changes in the eye reflexes, and when there is a well-marked ataxia as well as positive laboratory findings, it seems to me much more reasonable to ascribe the loss of function to a well-recognized cause rather than to the accident.

Case XII showed a characteristic symptom of nervous syphilis, and the laboratory findings were those of syphilis. The severity of the accident certainly did not suggest it as a cause of the paralysis.

Case XIII is included here since it represents a diagnostic problem which only time and treatment could answer. The complaint, dizzy spells, is purely subjective. The element in the history that smacked of exaggeration was the statement of the removal of the blood-clot. It seemed to me that the patient had some idea of clot on the brain, and expected me to understand

that the operation had to do with such a clot. From the location and character of the scar on the forehead an opening into the skull was out of the question. If the spells ceased soon after intensive antisyphilitic treatment was started it would be reasonable to ascribe them to syphilis. Otherwise, their nature would remain uncertain.

In Case XIV the patient was suffering from severe disease, probably muscular, of the brain stem. That the disability could be ascribed to the trifling injury of a month before was obviously ridiculous. However, this patient, in common with the other 13, had gone seeking damages.

I told you I would talk to you about disability, damages or disease. This I would amplify with the statement that when disability threatens there has to be damages or disease. If the injured doesn't feel that he has been given proper consideration, personal, medical, and financial, he keeps up his disability, takes it to court, and tries to capitalize it. He has to prove disease. If he is of a clever type, he will choose symptoms such as pain, dizziness, weakness, or loss of appetite. It is difficult to prove their presence or absence. The stupid man claims paralysis, blindness, or other loss of function, and falls into the trap with nothing but his own assertions to get him out.

Age plays an interesting part in the incidence of this condition. If a man of forty-five or over still in physical labor, suffers some relatively slight injury, but is brought to see thereby the spectre of physical disability threatening him, such a man is more apt to seek damages than one who is younger. His time to lay by something against the rainy day is short and he must take advantage of every opportunity.

In the remarks on symptoms I spoke of the sensory changes. There is one reaction that I have observed repeatedly. After jabbing the area claimed as analgesic for some time, if one touches the normal skin even quite lightly the patient will wince and otherwise overreact in a manner that seems to declare that he has stood pain just as long as he can, and as soon as he feels that he can do so he expresses the discomfort he has been feeling so long. When I see this reaction I feel certain of the patient's

dishonesty. As for the rest, the sensory changes are those of hysteria—the outlines shift, the analgesia is spread over an entire limb, stopping at axilla and point of shoulder, or at groin and great trochanter. Or it may be of a finger or of a hand, or run just up to the lower jaw from the collar-bone, or from the crest of the ilium to the lowest rib. I have often noticed that pricking of soft tissue is well borne, whereas equal irritation over bones—tibia, ribs, zygoma, etc.—is not.

It is surprising how often one of these men will say "No, no, no" as he is repeatedly touched over the anesthetic area, or will call each jab with the point of the pin "dull," and each touch with the head "sharp," or will say "down" each time the toe is moved up, and "up" each time it is moved down. Restrictions of the field of vision are popular symptoms. If the examiner will take the trouble to set the stage for himself he will readily bring out evidences of a lack of frankness. He can express expectations with change of the tone of voice, evoke expression of loss of sections of the field, and then prove to his own satisfaction that, in reality, no such loss exists. The same inconsistencies exist in the other functions. A man claiming partial hemiplegia will swing the disabled arm as he walks as no real hemiplegic does, or will expose use of the toes in walking—normal dorsal flexion as the foot strikes the floor—whereas if he is led to understand that free movements of the fingers and toes are not seen in hemiplegia, he will show an inability to perform free movements when they are asked for.

There is an almost endless variety of such signs and symptoms, but the essential element behind them all seems to be the lack of ingenuousness—they all show the desire to impress the examiner with an exaggerated idea of the disability. Thus, after all, is the diagnostic or pathognomonic element. And when it can be clearly demonstrated, no matter what other elements are also present, one can say that this individual wants damages and is going to try to convince the world that he deserves them.

CLINIC OF DR HARROLD A BACHMANN

FROM THE CARDIAC CLINIC OF THE CHILDREN'S MEMORIAL HOSPITAL

THE PREVENTION OF HEART DISEASE IN CHILDHOOD

CARDIAC disease has within the last several years established itself as a definitely important public health measure, and one which has received considerable comment in the medical literature, and more recently begun to invade the less scientific medical journals of the layman. The data thus submitted has been carefully obtained and analyzed, and upon review immediately impresses one with the vastness of the problem before us. The present status of our knowledge of heart disease as revealed by the literature can be readily summed up as follows: (1) That heart disease is tremendously prevalent both in children and adults, (2) that the outstanding etiologic factor is rheumatism and the other allied diseases, such as chorea and tonsillitis, (3) that actual cures are rather infrequent, but progression of the condition can be frequently abated providing intelligent care is given, (4) that the character and health of the cardiac muscle is of far greater prognostic import than is the integrity of the valves, (5) that our ultimate success lies along the lines of prevention.

Such is the essence of the problem today, and no doubt so it will continue to be for some time to come. It has seemed to me that these important points are at present quite generally understood by the vast majority of physicians and need no further emphasis. On the contrary, I feel that the application of the above points and the actual practical approach in the handling of cardiacs and the almost more important phase of preventing cardiac disease has been grossly neglected.

I find that almost the first question asked by interns and not infrequently by visiting physicians after a case has been demon-

strated, whether cardiac or otherwise, is, What can be done for it? Those of us dealing with large problems all too frequently become so engrossed in the bigger phases that we neglect to appreciate that the accumulation of statistics is not the all-important question to the man doing general practice. To him rather is it of interest to know of our results and how we obtain them. What our method is in dealing with the individual case and why.

In view of this fact I have thought it might be both interesting and instructive to show and review several typical phases of heart disease and its prevention as they occur in a children's cardiac clinic, attempting to describe our conduct in handling them and at the same time explaining our reasons for so doing.

Since chorea is generally more prevalent and rather frequently encountered in general practice, let us first proceed to the consideration of it.

Case I—This girl is eight years old. She has always been in good health except for occasional colds during the winter. At the age of five she had measles and the following year pertussis. There has never been any previous attack of chorea. In January, 1924 the mother noticed an increased nervousness and sensitiveness. The condition showed no improvement under home treatment, and she was brought to the clinic during the third week of her illness. She had by this time become more inco-ordinate in her movements and was unable to feed herself without encountering many accidents. There was twitching of the face and also some difficulty in speech. The diagnosis of a moderately severe chorea was made, and admission to the hospital advised. After a period of seven weeks complete recovery had occurred and she was discharged as cured. No cardiac involvement was found. Since this time she has returned to the clinic at regular intervals and continues to be classified as a potential cardiac, but has never shown evidence of heart disease.

Case II—This boy is now fourteen years old. At the age of seven he was admitted to the hospital with his first attack.

of chorea. He remained in the ward for two months and was discharged as cured with no cardiac involvement. Occasionally a soft basal systolic murmur was heard but was at all times considered as an accidental murmur. Since his first admission in 1917 he has returned to us with recurrences in 1919, 1922 and 1923. In 1920 he had his tonsils and adenoids removed. During his third attack of chorea in 1922 a harsh systolic murmur was discovered at the apex, which at the present time has developed into a definite presystolic murmur associated with a thrill. At no time has he had any rheumatic symptoms, nor has he ever been decompensated.

Case III—At the age of eight this girl was admitted to the ward with chorea, and after three months was discharged. No cardiac involvement was present. After six months of good health, she returned with the same symptoms, and following two months' stay was sent home as cured. On her third admission two years later with a chorea she complained of occasional joint pains and showed a definite arthritis involving the right wrist. At this time there was a systolic murmur of a blowing character at the apex, together with an accentuated second pulmonary sound. These findings have persisted, and today a diagnosis of mitral involvement can definitely be made.

DISCUSSION

In dealing with moderately severe choreas such as are seen in most dispensary services I find that when hospital facilities are available, admission is the most advisable procedure. Most of the homes from which these children come are far from adequate for the care of such patients. Likewise discipline is decidedly lacking. In private practice one does not as a rule, encounter quite such obstacles to hinder progress in the treatment of these patients.

Complete rest, exclusion of excitement, and good wholesome food still stand as the most specific treatment for chorea. Drugs are still used, and the one incidentally in use when the symptoms begin to subside receives the credit, while the hygienic treatment

noted above goes begging for its just reward. Except in the more severe types of the disease I have been impressed more and more with the inefficiency of drug therapy.

To the dispensers of Fowler's solution I might say I have yet to see any marked improvement in any case in which I have tried it. Likewise, the use of cacodylate of soda has never impressed me as of any great value. I still feel that any glowing result obtained from the use of either of these arsenical preparations is in direct proportion to the above hygienic treatment prescribed.

On the basis that chorea is a rheumatic manifestation, the salicylates have been frequently recommended. Whether they have a direct effect upon the invading organisms is still a question, but certainly in rheumatism we see definite clinical improvement. In chorea I have felt that the salicylates when administered in sufficiently large doses have given me the most gratifying results, and, in consequence, I have resorted to them more and more.

I have been using acetylsalicylic acid in preference to salicylic acid because it appears that larger doses can be given with less gastric disturbance. In children of eight years or older 15 to 20 grains can be given every four hours. I have occasionally given as high as 120 grains in twenty-four hours without observing any ill effects, though I feel that 75 grains are sufficient to produce results. Personally I am convinced that more uniform results are obtained with this medication than with any other.

In the most severe cases draw-sheets, both for the body and extremities, and at the same time luminal in doses of $\frac{1}{2}$ to 1 grain three times a day should be employed. Both these measures enforce rest and thus fulfil the requirements of the hygienic treatment.

Before I leave the subject of chorea I want to call your attention to these 3 children whom I have just shown, because I feel that they illustrate a classification which I find of value in determining the chances of subsequent cardiac involvement.

First The child who has only had one attack of chorea uncomplicated with any rheumatic symptoms. This child

almost invariably escapes heart involvement. If the parents are properly instructed as to the early symptoms of chorea, subsequent attacks can be averted and heart disease prevented.

Second. The child who has had frequent attacks of chorea. The likelihood of cardiac involvement increases with each repeated attack of the disease. For this reason direct your efforts always to preventing recurrences.

The third group is one over which you may have little direct influence. Here is included the chorea complicated with definite rheumatic symptoms, and in whom subsequent cardiac involvement is, with few exceptions, the result. Our efforts here must again be directed toward the prevention of recurrence of either condition, thus enabling the heart to return as nearly to normal as possible. Though I have not enough evidence at hand to quote statistics, I am convinced that this child, though presenting every evidence that the heart has been involved, may show in time neither sign nor symptom of heart disease. The process here may have been merely a mild inflammatory one without destruction or the formation of vegetations, and time and freedom from repeated infection have restored the valves and myocardium to at least clinical normalcy.

A recent study of the chorea cases on my service shows that about one-third of the children eventually show some degree of heart disease. In actual figures the above groups can well be illustrated.

Report of 162 Cases of Chorea

Without heart involvement	109
With heart involvement	52, or 32 per cent.

Of those with heart involvement there has been

Associated rheumatism	26
Frequent chorea	16
Frequent tonsillitis	10

This latter group forms a very definite one and introduces an etiologic factor in heart disease which must be considered seriously. In many cases frequent tonsillitis is the only factor in the history to which heart disease can be attributed, and when

added to it is the presence of a chorea, involvement of the heart should be anticipated.

Passing now to a consideration of rheumatic fever, it might be well to consider, first, one of the determining etiologic factors which lends great encouragement to the problem of heart disease from the standpoint of prevention. Evidence indicates as does a perusal of our files of private patients that rheumatic conditions are much less prevalent among the children of the well-to-do and those of more moderate circumstances than among the lower classes, both financial and intellectual. If we saw as much rheumatism and the subsequent cardiac involvement in private practice as we do in a dispensary of this type the incidence of heart disease in children would be markedly increased. No doubt there are several explanations for this discrepancy, but it seems to me it can be explained chiefly upon environmental influences both from a hygienic point of view and also from the fact that indispositions of childhood receive more prompt attention among the better class of parents.

This observation carries with it an extremely important point and one giving us a practical approach to our problem of heart disease prevention. It illustrates rather conclusively that efforts directed toward prevention are feasible. If healthful surroundings and indulgent care can keep our private practice surprisingly free of rheumatic cases, then surely the same measures instituted among the less fortunate classes will do likewise.

Regarding the symptoms of rheumatism in childhood there is only one point I wish to emphasize. The disease rarely manifests itself with the same degree of severity as it appears in the adult. Pain is present and not infrequently temperature, but the tender inflamed local signs are generally lacking. Likewise the articular form is not always present. Muscular pains of a mild variety, and usually considered casually as "growing pains," are not to be ignored. Until you have definitely explained a joint or muscle pain in a child as being other than rheumatic, in origin, consider it as such and treat the child accordingly.

Rather than take time to show you rheumatic cases, let me review some of the points which should be of special interest. Many of the children admitted will present a cardiac murmur which in the presence of the rheumatic symptoms prompt you to diagnose early cardiac involvement. This is rather an erroneous conclusion to draw, because in any acute infection with temperature in children such apical systolic murmurs can be heard. Real damage takes time to develop and these murmurs are due, rather, to muscular relaxation. They may indicate an invasion of the myocardium, but many of them are of a transitory nature, and with the subsiding symptoms disappear.

In a survey of the rheumatic cases on my cardiac list at present I find 82 cases. These do not include the ones still under treatment in the ward. Of the 82 children, there are 63, or 76 per cent, who show definite cardiac involvement. The damage varies from the mildest type to the most severe. The degree of cardiac damage is in direct proportion to the severity of the rheumatic conditions which preceded it. Thus, in the presence of rheumatic nodules, which invariably indicate a very extensive invasion, we anticipate, in consequence, an extensive carditis.

The treatment of rheumatism is quite generally known, but the rigidity with which it should be enforced is not fully appreciated. In the milder types I believe that the child, whether cardiac involvement be present or not, should be kept in bed for two weeks after the acute phase has subsided. In the more severe cases this should be extended. Because the pain and temperature have disappeared, let us not conclude that the infection is no longer present. I feel convinced that early activity following an acute rheumatic infection many times results in the subsequent cardiac damage which might otherwise have been avoided.

Rheumatism, like chorea, is subject to recurrences, and it is especially important to constantly emphasize this fact to the parents. Each recurrence adds to the likelihood of cardiac involvement. If it is not already present, and though we may not have had an opportunity to prevent the first attack, we do carry the responsibility of preventing subsequent attacks.

This brings up the question as to tonsillectomy, which at present is still a debated one, and probably will never be decided to the satisfaction of all. Removal of the tonsils is not a panacea, and if you approach the subject with that attitude, disappointment will be forthcoming.

It has always seemed to me that in childhood the nose and throat are the center from which most infectious processes have their inception. The common cold, or at least the signs and symptoms of this nature, precede most every condition we meet. This is almost invariably true in rheumatism and fairly frequent in chorea. If by the removal of these foci we can eliminate at least a proportion of these upper respiratory infections, something has been accomplished. Theoretically this sounds rational, and even though clinically the evidence is not always gratifying, I feel that the procedure is justifiable.

Finally, just a few words about the care of the child with established heart disease. The degree of damage may be estimated by the character of the heart tones and by the adventitious sounds produced, but of almost greater importance is an estimate of the functional capacity of that heart. More valuable information is obtained from a study of what the heart can do than from the sounds it may produce. The symptoms offer a better estimate of the heart's functional capacity than do the signs unless those signs give special evidence of myocardial involvement.

Thus our efforts should be directed toward the conservation of the myocardium, with the object at the same time of increasing its resistance and its efficiency.

First therefore, we must conserve it from extensive invasion during the acute infectious phase by the enforcement of rest until these acute symptoms have subsided. During this period, also, every means should be employed of increasing the general resistance and nutrition of the patient making at the same time the attempt to eliminate the original source of the infection, thus endeavoring to prevent recurrences. Whether tonsillectomy prevents rheumatism or not no conclusive evidence has been produced but clinically I feel inclined to believe that tonsillec-

tomy does lessen the frequency of additional cardiac trouble after lesions have once been discovered

Our second efforts should aim at establishing again a cardiac muscle as nearly normal as possible, with as wide a power of reserve as it will permit. The first measure will increase its resistance against further invasion, while the second will protect against decompensation should subsequent invasion take place.

Exercise and normal play are today considered essential in the treatment of heart conditions, and through their intelligent use is increased the health and tonicity of the myocardium. Certain restrictions and limitations are, of course, necessary, and, in general, those activities involving competition and tests of endurance. The extent to which exercise is indulged in must be regulated entirely by a close study of its effect upon the individual heart in question. Cardiac decompensation as seen in children is rarely due to exercise, providing the acute infection has subsided. Exercise when properly supervised is in itself a most valuable adjunct to the proper care of a cardiac patient.

The third factor to be considered, as in the previous conditions mentioned is to direct an unceasing fight against additional infection. Prevent fatigue by establishing a normal routine of rest and relaxation such as even a normal healthy child needs during that period when growth and development are at their height.

Likewise establish an appetite, not with tonics, but through education and habit. Most of the poor eaters I see in practice are poorly disciplined children. The desire to eat in a child is as normal a function as the desire to urinate but too often is permitted to become distorted and perverted by overindulgent parents.

Finally, but only with the support of the above, we must endeavor to eliminate the most common and at the same time the most important of all human ailments—the common cold. In it lie all the potentialities of most of the serious medical conditions observed in childhood. Insignificant as it may be in the normal child at most times to the child with a cardiac lesion

it is always a dangerous menace. Almost without exception the cases of decompensation seen in cardiac children occur as the end-result of one of these generally considered benign nose and throat colds. If, however, your resistance has been increased, together with a constantly improving myocardium, progression of the cardiac condition can be averted and a useful life assured.

CLINIC OF DR WILLIAM H HOLMES

WESLEY MEMORIAL HOSPITAL

CASES ILLUSTRATING THE SYMPTOMATOLOGY OF BRAIN-STEM LESIONS AND OF CHRONIC SUBDURAL HEMORRHAGE

Case I Amyotrophic Lateral Sclerosis

Case II Occlusion of the Postero-inferior Cerebellar Artery

Case III Tumor of the Cerebellopontine Angle Arising from the
Auditory Nerve

Case IV Compression Tardive (Pachymeningitis Hæmorrhagica
Internæ or Chronic Subdural Hemorrhage).

THE recognition of the nature and location of morbid processes in the central nervous system is of the greatest importance As a truism this statement needs no support The following cases, therefore, have been chosen for presentation because they illustrate various aspects of interest to internists, neurologists, and surgeons

CASE I AMYOTROPHIC LATERAL SCLEROSIS

The patient, M R, a white woman, aged fifty-nine years, referred by Dr Thomas G Jones, dates her present illness from an injury sustained sixteen months ago In March, 1924 she fell and struck the right breast There was discoloration, swelling, and pain After the signs of local injury had subsided she noticed that she became fatigued easily In September she began to have pain in the right thumb A short time thereafter she was experiencing pain in the entire hand Within two months the right elbow and shoulder were involved The pain, especially that in the shoulder, was described as being very severe and

aggravated by motion. Associated with the pain there was a gradually increasing weakness of the hand, arm, and shoulder. As the muscular weakness increased, stiffness of the muscles and fixation of the joints increased *pari passu*. When fixation of the joints had become complete the pain disappeared. In April, 1925 she consulted a neurologist, who had her admitted to a hospital for study. The history and progress notes made at that time have been placed at my disposal. It is recorded that the general medical examination was negative. Laboratory tests contributed little. The impression was that of a brachial neuritis. There was atrophy of the right hand, arm, and forearm. The hand was cold and cyanotic and it is recorded that sensation to both touch and pain in the distribution of the right ulnar nerve was reduced. No evidence of disease of the central nervous system was found, unless a statement that there was spasticity of both arms can be regarded as such. That the process was bilateral is borne out by the present physical findings. The severe pain, muscular atrophy, and sensory diminution in the distribution of a peripheral nerve formed the basis for the diagnosis of brachial neuritis. It is of interest to recall that the pain did not involve the entire extremity at any one time. It began in the most distal segments and ascended to the forearm, arm, and shoulder during a period of two months, and was first experienced six months after the injury to the right breast. No foci of infection were found nor were there any evidences of intoxication. It is extremely doubtful whether an injury limited to the mammary gland could be responsible for a brachial neuritis in the absence of a history of pre-existent infection or intoxication. In any event, the diagnosis of neuritis, in the limited sense in which the word is ordinarily used in referring to a purely peripheral disease of the nervous system, is no longer applicable to this case. At the present time there is no complaint of spontaneous pain, but pain can be produced by attempting to move the rigidly fixed shoulder. It is evident that this is caused by efforts to overcome the contractures, and is similar to the pain experienced by patients on first trying to use an extremity which has been immobilized for weeks or months.

Physical Examination (July 15, 1925).—The general appearance of the patient is pathetic in the extreme. She is much underweight, due very largely to difficulty in swallowing. Food accumulates between the gums and cheeks and cannot be dislodged by the tongue. Liquids induce attacks of coughing and are often regurgitated through the nose. She has difficulty in swallowing saliva. The face lacks expression, the mouth is kept partly open. There is constant fibrillary trembling of the orbicular muscles. The tongue is protruded only as far as the margin of the lips, it remains centrally placed and cannot be moved to either side, it is reduced in volume and has a flabby appearance. Speech is almost unintelligible even to her son and attendants. It can best be imitated by attempting to speak without using the tongue. Hearing, vision ocular and jaw movements, and facial sensation are perfectly normal. The only cranial nerves involved are those supplying the tongue, lips, pharynx, and larynx, giving rise to the so-called glossolabio-laryngeal paralysis or bulbar palsy. Mentally and emotionally the patient is quite normal. There is advanced atrophy of the entire right arm and a lesser degree of atrophy of the left arm. Associated with this pronounced atrophy there is a muscular hypertonicity as evidenced by the rigidly fixed position and the greatly exaggerated tendon reflexes. The lower extremities show no signs of atrophy, but muscular hypertonicity is evident. The knee- and ankle-jerks are exaggerated; there is definite left ankle-clonus and a suggestion of ankle-clonus on the right. Passive movement and walking further confirm the finding of spasticity. The superficial abdominal reflexes are present, but are obtained only with difficulty after repeated trials. There is normal plantar flexion on the right side. On the left there is a typical Babinski sign. Vesical and rectal control is not impaired. Pain, tactile and vibration sensibility of the entire body is normal. There is no evidence of ataxia of the lower extremities. Rigidity prevents testing of the upper extremities.

The findings are exclusively in the sphere of motor control and indicate involvement of both the upper and lower motor neurons. The extrapyramidal tracts apparently have been spared.

Discussion—Atrophy without sensory loss excludes disease of the peripheral nerves and indicates a lesion involving the anterior horn cells of the cord and the motor nuclei of the medulla. Were these the only cells affected the atrophy would be associated with loss of muscle tone and decreased or absent reflexes in the muscular distribution of the diseased cord segments. Such are the findings in anterior poliomyelitis, in which groups of muscles may be paralyzed. In this disease contractures occur, but the contractures are due to the unopposed action of healthy muscles. Here, the atrophy indicating involvement of the lower motor neuron with its specialized function of idiodynamic control of the muscles is associated with hypertonicity of the paralyzed muscles, with exaggerated tendon reflexes throughout, with ankle-clonus with decreased abdominal reflexes, and a typical Babinski sign indicating involvement of the upper motor neuron. Retention of pain sensibility in the atrophied area excludes syringomyelia. The absence of nystagmus, disk changes and tremor excludes multiple sclerosis. The hypertonicity excludes chronic anterior poliomyelitis. The association of atrophy, hypertonicity, bulbar palsy and retention of all forms of sensation characterizes a rare, progressive, and fatal disease. The diagnosis is amyotrophic lateral sclerosis. The advanced bulbar palsy indicates an early fatal termination. The most probable cause of death in this stage of the disease is aspiration pneumonia.

CASE II OCCLUSION OF THE POSTERO-INFERIOR CEREBELLAR ARTERY

The patient a white man, aged forty years, a physician engaged in general practice, is referred for diagnosis by Dr. J. A. Brown.

He complains of inability to control the movements of his right leg. The onset of this disability began acutely three weeks ago, and was accompanied by other symptoms which are described very clearly by the patient.

He has had unusually good health. He recalls one illness a few years ago lasting six weeks, which was diagnosed as acute rheumatic fever. At that time he had pain in the ankles, fingers

and wrists, but there was little if any fever and no sweating. He attributes the illness to a streptococcal infection, although he did not have a sore throat, nor was there any cardiac complication or anemia. This illness may have been acute rheumatic fever even though it lacked some of the characteristic features of that disease, but there is a possibility that it was a metabolic disturbance of some kind like an atypical attack of gout. The patient is married and has 2 healthy children. Venereal disease by name and symptoms is denied. His habits are fair, he smokes cigarettes and uses alcoholic liquors in moderation. He is not subject to headaches, vertigo, dyspnea.

On two occasions, about four weeks ago, he had pain in the right eye which lasted a few seconds. One week later, just after breakfast, as he was about to start on his professional rounds, he had a stroke of intense neuralgic pain behind the right eye. Simultaneously he became very dizzy and felt as though he was falling to the right. There was a sensation of choking, as though there was an obstruction in the larynx. The vertigo was not associated with tinnitus and there was no disturbance of vision. While lying in bed vertigo was not present, but it returned immediately on raising his head. The symptoms of pain, vertigo, and choking were followed by numbness of the right side of the face. Examining his face in a mirror, he observed that the right pupil was smaller than the left and that the palpebral aperture was narrowed. About two hours after the onset of symptoms he vomited for the first time. Vomiting recurred several times the first day, but not thereafter. He was mentally quite clear, but felt very drowsy. He next noticed that over a limited area of the left side he was unable to appreciate the temperature of the water with which he was being bathed. Touch sensibility seemed to be perfectly normal in this area, but on pulling out hairs he did not experience pain. By this means he was able to map out an area of analgesia on the left side, beginning at the costal margin, including the abdomen, lumbar region, buttocks, scrotum, and leg. About the time he detected analgesia of the left leg he experienced difficulty in controlling the right leg. On drawing the leg up in bed it would

invariably fall into a position of abduction and external rotation. He remained in bed for ten days. Vertigo on raising the head gradually became less severe and finally ceased, the numbness of the face and analgesia of the left leg disappeared but the gross clumsiness of the right leg remained, so that the use of a cane was necessary in walking.

Physical Examination Three Weeks After Onset of Symptoms—The patient does not appear to be acutely ill. He is up and about, has a normal pulse and respiratory rate. Temperature normal. Systolic blood-pressure 195, diastolic pressure 100. Aortic second sound accentuated. Urine normal. Wassermann test on the blood negative. Spinal fluid not examined.

There is scarcely a detectable weakness of the right side of the face, i.e., the palpebral aperture is slightly narrower than the left and the angle of the mouth droops. The right pupil is smaller than the left, but both are regular in outline and react promptly to light and accommodation. Function of the external ocular muscles is normal. Using the Maddox rod, there is perfect fusion of images. Nystagmus is not induced by changing the position of the head. The visual fields tested roughly by the moving finger method are normal in shape and size. The media are clear and the vessels and disks normal. Jaw movements are normal and there is no disturbance in the facial innervation. Hearing of the spoken and whispered voice is normally acute. Bone and air conduction tested with a Cl tuning-fork gives normal reactions. Pharyngeal reflexes are normal. Tongue is protruded in the median line without tremor. Test sentences are repeated without error and spontaneous speech is normal. Bárány tests not made.

Motor System—Flexion and extension of the forearms and grasp of the right hand are slightly diminished. Rapid pronation and supination of the hands are performed without difficulty. Tests for spontaneous past-pointing are negative. There is no tremor, astereognosis, nor rebound phenomenon, writing is normal. The right lower extremity shows a slight degree of weakness when compared with the left. Tests for ataxia are well performed. The automatic movement of walking, however, demonstrates difficulty in controlling the movements of the right leg. The leg

is thrown outward and forward. The asynergy is not increased by closing the eyes.

Reflexes—The tendon reflexes are brisk and equal. The superficial abdominal and cremaster reflexes are present and equal. Gordon, Oppenheim, Chaddock, and Babinski signs are absent.

Sensation—Sensibility of the face, trunk, and extremities tested with a wisp of cotton for tactile sense, and with a pin for pain sense is normal. Vibration sense and muscle and joint sense are normal.

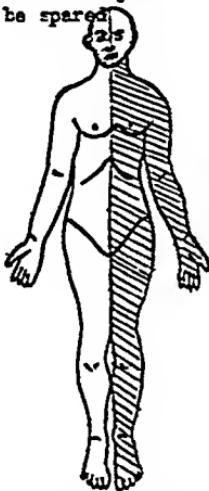
Loss of all types of sensibility although tactile may be spared.

Nystagmus

Horner's syndrome

Laryngoplegia

Hemiasynergia
w. th. vertigo and
falling to the right
Tremors
Normal sensation
Organic reflexes
normal.



Loss of pain and temperature sense. Face spared.
Tactile and deep sensibility normal.

Fig. 270.—The typical clinical findings in occlusion of the postero-inferior cerebellar artery.

Discussion—Cases I and II had this feature in common, the morbid processes involved the medulla in both. In one, the pathologic process was slowly progressive, involving only the motor nuclei. In the other the onset was acute, the symptoms alarming, and motor and sensory tracts were involved in a most bizarre manner. It is evident that the pathologic conditions were entirely different, the one was degeneration, the other, unquestionably, a vascular accident. Were the symptoms in Case II due to embolism, thrombosis, hemorrhage or the pressure of an aneurysm? The existence of hypertension suggests the

possibility of hemorrhage. There was no evidence of cardiac disease, and, therefore embolism is a remote possibility. The postero-inferior cerebellar artery supplies the area involved. Occlusion of this vessel gives rise to a very characteristic group of symptoms subject to some variation, depending on the degree of occlusion. The typical clinical findings of complete occlusion are illustrated in Fig. 270.

The postero-inferior cerebellar artery is the largest branch of the vertebral. It arises a short distance below the pons, and passes obliquely backward around the lateral surface of the medulla, which it supplies by small branches. The deeper central and the ventral parts of the medulla are supplied by branches of the anterior spinal artery. The mesial fillet and the pyramids, therefore, escape injury in occlusion of the posterior inferior cerebellar artery. The structures most frequently involved are: The formatio reticularis, the descending sensory root of the trigeminus, the nucleus ambiguus which gives rise to important fibers of the vagus, the glossopharyngeal nucleus, the spinocerebellar tracts, the vestibular nuclei, the inferior cerebellar peduncle, and the spinothalamic tract. The majority of these structures are placed superficially on the lateral aspect of the medulla (Fig. 271). If deeper and more centrally placed structures, such as the mesial fillet, the hypoglossal nucleus, or the pyramids are also involved, or if pontine symptoms exist, occlusion of the vertebral artery is indicated. Occlusion of the vertebral is, therefore, indicated by involvement of structures in addition to those involved in occlusion of the postero-inferior cerebellar artery.

In the case under discussion the sudden severe pain behind the right eye indicates an irritative lesion of fibers in the sensory root of the trigeminus nerve. The course of these fibers is indicated in Fig. 272. The numbness also indicates interference with the function of these fibers.

The complaint of laryngeal obstruction indicates involvement of one of the nuclei of the vagus—the nucleus ambiguus. The dorsal nucleus of the vagus which controls cardiac, respiratory, and gastro-intestinal activity, was not affected.

The vertigo indicates involvement of the vestibular nuclei. The loss of pain and temperature sense of the lower part of the

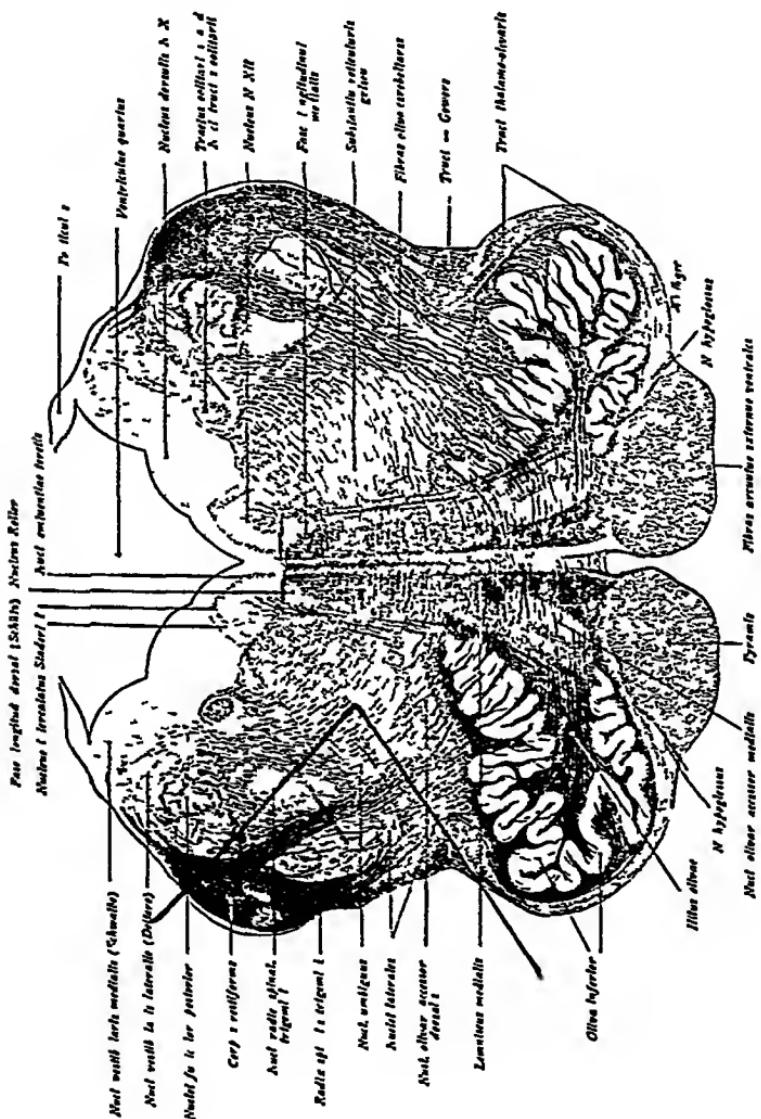


FIG. 271 — Illustration from Villiger to show the area supplied by the postero-inferior cerebellar artery.

left side of the body indicates involvement of the most superficial fibers of the spinothalamic tract. This tract is also called the spinal fillet because the sensory fibers which enter into its forma-

tion decussate almost immediately after entering the spinal cord. In the typical syndrome following occlusion of the postero-inferior cerebellar artery deep sensibility is not impaired because the fibers conveying this type of sensation ascend in the posterior columns to the nuclei of Goll and Burdach in the medulla.

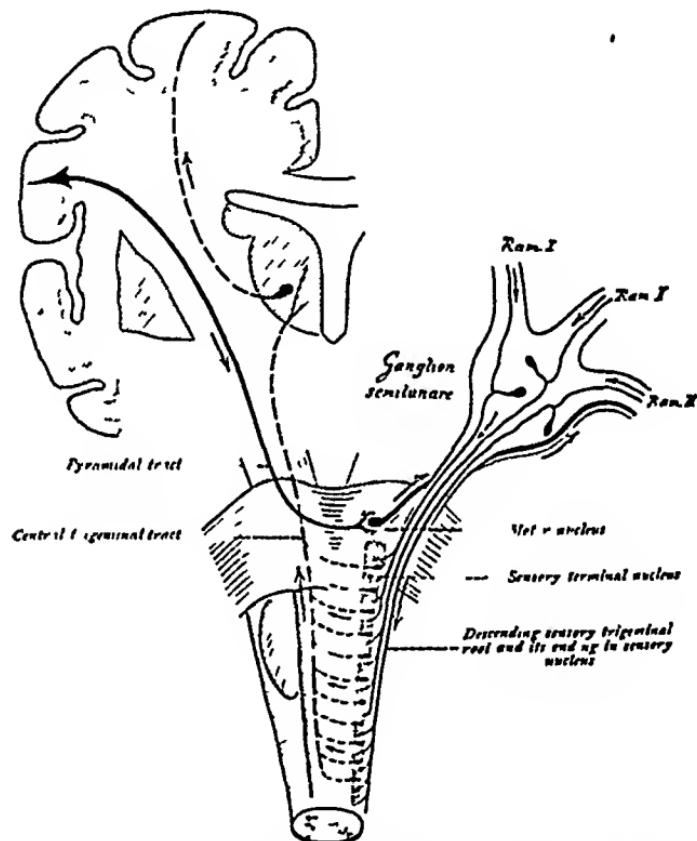


Fig. 272.—Illustration from Villiger to show the course of the sensory fibers of the trigeminal nerve.

From these nuclei fibers arise that decussate in the mesial fillet which receives its blood-supply from the anterior spinal artery.

The asynergy of the right leg is attributed to involvement of the spinocerebellar tracts.

The myosis, narrowed palpebral aperture, and drooping of the angle of the mouth constitute an incomplete Horner's syn-

drome which is attributed to involvement of the central connections of the sympathetic system Horner's syndrome does not always indicate central disease, it is often caused by injuries to the sympathetic trunk or ganglia in the neck from stab or bullet wounds or as a result of pressure from tumors or goiters

Unilateral irritation of the sympathetic nerves in the neck causes homolateral symptoms consisting of dilatation of the pupil, exophthalmos, widening of the palpebral aperture, and sweating of the face

Compared with the frequency of vascular accidents in other parts of the central nervous system, occlusion of the postero-inferior cerebellar artery is a rare occurrence, G W Robinson, reporting a case in 1913, collected 28 cases from the literature Since then additional reports have appeared by Hall, Hausman, Hammes Fisher, Gillis, Anderson, and one or two others As already stated, the symptoms vary with the degree of medullary involvement, but vertigo, facial anesthesia, asynergy, and some degree of contralateral analgesia and thermo-anesthesia are essential for a diagnosis In only a few of the cases were the symptoms ushered in by neuralgic trigeminal pain Wallenberg's case had pain in the left eye at the beginning of the symptom complex Thomas' case also had initial pain Often the vertigo is increased by movement and decreases or disappears while the patient is lying in bed Bárány tests have been recorded in the cases of Hall and Fisher It is surprising to note how many of the reported cases have recovered, so that the extent of the medullary damage could not be determined Those interested in the pathology will find the article by Spiller particularly helpful

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CASE III TUMOR OF THE CEREBELLOPONTINE ANGLE ARISING FROM THE AUDITORY NERVE

Vertigo is an extremely common symptom caused by a great variety of conditions. It may be the result of a vascular accident in the medulla. It may also be caused by a simple arteriosclerosis without evidence of hemorrhage, thrombosis, or embolism. It occurs in toxic states of both exogenous and endogenous origin. It may be a symptom of anemia or of polycythemia, of aortic insufficiency, of degenerative nervous diseases, of otitic disease, and of tumor of the brain. It is commonly seen in pregnancy as a prodrome of epilepsy and as the result of paralysis of ocular muscles. There are many very normal people who are unable to ride any considerable distance on a street car or train because of vertigo. The stewards on lake and ocean steamers are well aware of the ease with which unusual stimuli may upset man's equilibratory apparatus. Vertigo should be differentiated from mere faintness. Patients very frequently state that they have attacks of dizziness when what they mean is that they feel faint. The following history illustrates the occurrence of vertigo in a case of tumor of the acoustic nerve.

A P., a white woman, aged thirty-eight years, referred for diagnosis by Dr. W. S. Boughey.

History — In August, 1921, while in the yard hanging up clothes, she experienced her first attack of vertigo and of spasm of the left side of the face, which lasted three or four minutes. During the period of vertigo external objects seemed to rotate from left to right. Immediately following the attack of vertigo she fell asleep and slept for about thirty minutes. On awaking she resumed her work and was not conscious of any unusual sensations. Prior to this she had been in good health.

August 18, 1921 she became pregnant for the third time. In two previous pregnancies she had vomited during the first three months, but in this pregnancy she vomited throughout the entire period of gestation. She did not suffer from either vertigo or facial hemispasm, although nausea and vomiting were so annoying. Pregnancy terminated normally May 23, 1922, although for some weeks there had been albuminuria and hypertension.

On July 3d her systolic pressure was 200, on July 15th, 185, on July 30th, 165. Sometime during July, about six weeks after the birth of her child, she had her second attack of vertigo and facial hemispasm. This attack was similar to the other, even to the extent that it also occurred while hanging out a washing of clothes. A short time later the third attack occurred while reaching for an electric light chain pull. Thereafter attacks occurred very frequently, at least twice a week. The patient's husband witnessed a number of attacks. In his opinion vertigo occurred only when she changed the position of her head. The vertiginous attacks at first were always accompanied by facial hemispasm, but later vertigo occurred without spasm. During the attacks of vertigo which occurred under all sorts of conditions, except that no attack occurred while turning in bed, she felt that external objects rotated to the right while she had the sensation of remaining stationary. She also stated that putting the head back not only caused vertigo but also resulted in the production of a sound as of a water-fall. Some time during the course of her pregnancy she noticed that she was deaf on the right side, and about the time her pregnancy terminated she complained of failing vision.

Physical Examination (September 11, 1922) — Eyes Conjunctiva and sclera were normal. There was no ptosis or paralysis of the extra-ocular muscles. No exophthalmos. A marked horizontal nystagmus with the quick component in the direction of vision was easily induced. On looking downward a rotatory nystagmus was induced. No nystagmus on looking upward. The pupils were 3 mm. in diameter, equal in size, regular in outline, and reacted promptly to both light and accommodation. The consensual and ciliospinal reflexes were normal. Ophthalmoscopic examination showed a choking of both disks of about two diopters. Visual fields for form were normal in size and shape.

Sensation of the face and motor function of the fifth nerve were normal on both sides. Taste sense on the anterior part of the tongue was normal. There was no asymmetry of the face. There was no disturbance in the facial innervation.

Ears Inspection of the external ear showed no evidence of disease. Neither whispering nor speaking was heard on the right side. Normal hearing on the left side. A tuning-fork placed on the vertex was referred to the left ear. When placed over the mastoid on the right side it was heard for twelve seconds. (This was probably due to conduction through the bone to the left ear.) With a Bárány noise machine in the left ear the right ear was found to be totally deaf. Bárány caloric and turning tests, performed by Dr C. F. Bookwalter, demonstrated a complete absence of vestibular reactions on the right side, with normal reactions on the left side.

The tongue was protruded in the median line without tremor. There were no disturbances of speech, swallowing, head turning movements, etc.

Reflexes Conjunctival reflexes were present and normal. All superficial reflexes, including the plantar, were normal. The tendon reflexes on the right side were slightly less brisk than those on the left. Motor function was normal. The finger-to-finger and finger-to-nose tests were well performed. Rapid pronation and supination showed no evidence of cerebellar disease. Rebound phenomenon absent. With the eyes shut the patient was always able to find the finger of the examiner and could point to various objects in the room without error.

Station On standing erect there was unsteadiness, with a tendency to lean toward the right and slightly backward.

Gait There was an unsteadiness of gait, with slight swaying from side to side. Forward and backward walking with the eyes closed did not increase the swaying perceptibly, nor was there any constant tendency to turn on her own axis. Sensation of the trunk and extremities was normal.

Pulse rate 84, systolic pressure 130, and diastolic pressure 90.

Blood Wassermann negative to two antigens. Spinal fluid not examined. R. B. C. 5,148,000. Hemoglobin (Dare) 90 per cent. W. B. C. 8200. Urine negative except for a very faint trace of albumin. Blood creatinin 1.2 mgm per 100 c.c. X-Ray plates of the skull were negative.

Summary—Bilateral papillitis Loss of function of the right cochlear nerve Loss of function of the right vestibular nerve Hemispasm of the left face Vertigo

Decreased tendon reflexes on the right side or increase on the left Romberg, with swaying to the right and backward

The following report was made "The history and findings, in my opinion, indicate that the morbid process is both extra-cerebellar and extrapontine It is apparently a tumor in the right cerebellopontile angle and is a neurofibroma arising from the right acoustic nerve "

Operation was advised, and on October 11, 1922 the patient was admitted to Wesley Memorial Hospital On October 13th Dr D C Good, the House Physician, observed an attack of facial hemispasm not associated with vertigo He stated that the right eye was kept open, the left closed, the mouth drawn to the left side and that the head was turned to the left The spasm lasted less than a minute and was followed by flushing and sweating of the face, most marked on the right side Operation was performed by Dr A B Kanavel on October 16th A tumor mass 2 cm in diameter was found in the right cerebellopontine angle It was removed *en masse* Following its removal there was considerable oozing of blood A cigarette drain was placed down to the site of the removed tumor Section of the tumor proved it to be a fibroma The day following operation the patient's condition was very satisfactory No additional cranial nerves had been injured

The diagnosis of tumor of the right eighth nerve having been confirmed by operation, there are one or two points in the history which deserve some comment The characteristic symptoms of cerebellopontile angle tumors are generally known and are easily recognized Since the tumor arises from the auditory nerve, nerve deafness is an early and essential symptom The deafness may be complete, however, without the patient being aware of any impairment in hearing Tinnitus may be present, but is not an essential symptom Involvement of the vestibular portion of the eighth nerve gives rise to vertigo which may be regarded as the second essential symptom The next group of

symptoms is caused by pressure on the pons and cerebellum. The fifth and seventh nerves on the same side may be involved, giving rise to homolateral numbness of the face and to paresis or spasm of the facial muscles. Cerebellar involvement is indicated by an inability to harmonize the actions of muscle groups on the side of the lesion. The gait is the typical cerebellar ataxia with a tendency to fall toward the side of the lesion.

By the time the tumor has grown sufficiently to produce pontine and cerebellar symptoms obstruction to the flow of cerebrospinal fluid occurs, with the production of secondary hydrocephalus and a general rise in intracranial tension. All of these symptoms with the exception of involvement of the fifth and seventh nerves were present in the case of A. P.

In addition, there was an atypical symptom, namely, spasm of the face *on the side opposite* to the lesion. The last recorded spasm occurred before removal of the tumor. Spasm of the face *on the side of the lesion* has been observed in other reported cases and is easily explained.

Cushing, in his monograph on Tumors of the Nervus Acusticus, says "Irritative symptoms referable to the facial have been more frequently recorded than is generally supposed."

"Stewart and Holmes mention them as possible, though inconspicuous, symptoms. They were described, we may recall, by Cruveilhier as conspicuous features of his case, and in 1865 Hughlings Jackson noted contralateral attacks of facial spasm which he had difficulty in accounting for. They occurred in Brückner's, Sorgo's, and in Alexander and Frankl-Hochwart's patients, two examples are cited by Mills and Weisenburg, and some slight facial twitching was present in 3 patients in these series, in one of them on both sides of the face".

The second point which deserves comment is the entire absence of vertiginous attacks during pregnancy, and their reappearance shortly after the termination of pregnancy. Cushing and others have observed similar cases.

The postoperative course during the first four days was not only satisfactory, but very encouraging. The subsequent

course was very stormy. The progress notes are of some interest and excerpts will be quoted.

October 18th "Speech is very thick and almost unintelligible. Nystagmus unchanged. Subjective vision better. General condition good, but not good enough to permit a thorough neurologic examination. Both cigarette drains removed."

October 28th "There is clouding of consciousness. Not well oriented. There is slight paresis of the right side of the face. Saliva flows from the mouth constantly. Marked difficulty in phonation in part due to difficulty with labial sounds. Pulse rate around 120 most of the time. Horizontal and vertical nystagmus present. There seems to be a weakness of the right external rectus. Slight exophthalmos of the right eye. Involvement of the right sixth, seventh, eighth, ninth, and tenth cranial nerves."

November 1st "Patient's mentality still below normal, but is somewhat brighter today than she has been. Her urinary output has been far below normal and her general condition seems more suggestive of a renal condition than anything due to her operation."

Examination of the blood showed the presence of 50 mgm of non-protein nitrogen, 15.9 mgm urea nitrogen, and 3.5 mgm of uric acid per 100 c.c. of blood.

November 9th "Patient still clouded. Does not co-operate well. Refuses nourishment. Speech unintelligible jargon. Right sixth and seventh nerve paresis unchanged. Slight horizontal and vertical nystagmus. Reclines most of the time on the right side. It is impossible to test movement satisfactorily, but it is noted that the left hand is used more than the right. On reaching for objects with the left hand there is marked asynergy. Constantly overreaches. Right hand shows even coarser movements, but is not used voluntarily. Bulging on the right side of occiput at site of operation."

The patient was discharged December 14th. The hernia mentioned in the progress note of November 9th was still present. The mental state and the evidences of medullary and cerebellar compression were not improved at the time of discharge. Death

occurred suddenly on December 16th. An autopsy was refused. The embalmer reported that injection of embalming fluid into the vessels resulted in a very rapid accumulation of fluid in the tissue of the neck on the right side. This would indicate that the bulging noticed during life was due to an accumulation of blood. Evidently there was either oozing or intermittent bleeding after the removal of the cigarette drains. A second operation might have relieved the medullary compression, but whether permanent hemostasis could have been effected is questionable. The surgical removal of tumors in the cerebellopontile angle is attended with unusual difficulties. Cushing advocates removal of the tumor piecemeal if necessary, leaving the capsule *in situ*. He has found that the danger of hemorrhage is materially decreased by this conservative attitude.

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CASE IV COMPRESSION TARDIVE (PACHYMENINGITIS HÆMORRHAGICA INTERNA OR CHRONIC SUBDURAL HEMORRHAGE)

A. C., a white man, aged forty-seven years, was seen in consultation at the Englewood Hospital with Dr. W. R. Abbott, the evening of January 6, 1925. The family and previous personal history will be omitted because they have no bearing on the case. The history of the injury which led to his admission to the Englewood Hospital is as follows:

While on his way to work the morning of December 23, 1924 he slipped on an icy sidewalk and fell, striking his occiput on the pavement. Immediately after the accident there was bleeding from the nose for a few minutes. He felt dazed and complained of headache. Thinking that the headache would disappear he reported for work as usual and worked until noon. The headache instead of abating became more severe and was felt in the temporal and frontal regions. There was considerable vertigo. On arriving at his home he vomited. There was no further vomiting throughout the duration of his illness. On December 27th, the fifth day of illness, he was seen for the first time by Dr. Abbott,

who made a diagnosis of skull fracture and urged hospital care, which was refused. He was not seen again until December 29th, the seventh day of illness when he consented to enter the hospital for observation and treatment. He was still complaining of severe headache and of dizziness. There was no disturbance of speech, consciousness, or of motor functions. A-Ray study of the skull by Dr W R Bronson showed a faintly visible linear fracture of the occipital bone. He was given sedatives, and in two days he felt so well that he insisted on being up and about, although he still complained of headache.

January 1st, the tenth day of illness, it was noticed that his speech was thick and indistinct. During the day he slowly became unconscious and remained in this condition for a period of four hours. On regaining consciousness he appeared to be perfectly clear. Temperature, pulse, and respiration were normal. There had been no evidences of a convulsive seizure. Speech continued to be indistinct, but he was not aphasic. From January 1st to January 6th periods of consciousness with sluggish mental reactions alternated with gradually lengthening periods of coma.

Physical examination at 5:30 P.M. January 6th the fifteenth day of illness. Patient was in deep stupor, did not respond to questions or commands, resisted examination.

Heart, lungs, abdomen, urine, etc., negative.

The pupils were equal in size, regular in outline, contracted, and reacted to light stimulation. There was no nystagmus and no evidence of palsy of any of the extra-ocular muscles. A fairly satisfactory ophthalmoscopic examination showed no evidence of choked disk.

The face was symmetric. The tongue and pharynx were negative.

Motor There was hypertonicity of the muscles of the left side. Power of the muscles of the left arm and leg was diminished. This was determined by his resistance to examination.

Reflexes Tendon reflexes of the left arm and leg were slightly more active than those on the right. There was no clonus. The superficial abdominal reflexes on the left side could not be obtained, while those on the right side were brisk. The

left cremaster reflex was sluggish, the right brisk. There was normal plantar flexion on both sides.

Spinal puncture. The fluid flowed drop by drop. There was no turbidity. Nonne and Noguchi tests were negative. There were 9 cells per cubic millimeter, of which 6 were red blood-cells. The Wassermann and Lange tests reported subsequently were negative.

Diagnosis.—On the basis of the history of injury and the x-ray findings a diagnosis of fracture of the occipital bone had been made. On the basis of the neurologic findings this diagnosis was enlarged to include increased intracranial pressure due to encysted hematoma in the right parietal region. Operation was advised. At the time this diagnosis and recommendation was made an explanation of the following points was requested:

1. If the coma is due to hemorrhage in the right parietal region, why did it not appear on the first or second day after the injury instead of the tenth?

2. If there is a sufficient extravasation of blood in the subdural space to produce coma, how can the absence of blood from the spinal fluid be explained?

In the majority of cases of skull fracture the symptoms of intracranial hemorrhage make their appearance very early and become progressively more threatening. Examination of the spinal fluid usually shows the presence of blood.

In this case headache and vertigo followed the injury immediately, but there was no loss of consciousness until the tenth day. Thereafter periods of consciousness alternated with periods of stupor or coma. The spinal fluid was in no way abnormal, the red cells being so few that their presence might easily have been caused by the puncture. These facts by no means exclude intracranial hemorrhage. They do, of course, exclude the ordinary type of hemorrhage in which a relatively large amount of blood is forced into the subdural space within a few seconds. There is, however, a type of hemorrhage usually following minor injuries in which the blood escapes very slowly and becomes encysted. The difference in symptomatology may be due to the rate and volume of the extravasation. The encapsulation of the effused

blood explains the absence of red cells from the spinal fluid. The correctness of the diagnosis was confirmed by operation the following morning. The surgeon, Dr Abbott, laid down a large osteoplastic flap in the right parietal region. On opening the dura an encysted collection of blood was found and removed. The patient made an uneventful recovery.

Speaking of this type of hemorrhage, Purves Stewart says "Another important form of cerebral hemorrhage which produces cerebral compression is *chronic subdural hemorrhage*, or "compression tardive," in which a collection of blood, often very large, gradually accumulates between the dura mater and the cerebral hemisphere. This blood is enclosed within a distinct membrane derived from the coagulated blood itself. Its longest diameter, which may measure 4 to 6 inches, is usually antero-posterior, most commonly in the frontoparietal region. The condition is sometimes bilateral. The bleeding is venous in origin and is due to rupture of the short cerebral veins which enter the superior longitudinal sinus almost at right angles. These veins are firmly fixed at one end in the rigid dura, while they are attached at their cerebral end to the relatively movable brain. Chronic subdural hemorrhage is the result of a trauma, but the injury is generally comparatively trivial, so much so, that unless inquired for, it may be entirely overlooked. A sharp blow, especially on the front or back of the head, may, without any fracture of the cranium, or even without a scalp wound, suddenly dislocate the hemispheres and rupture the veins on one or both sides. The resulting hemorrhage is a slow or intermittent venous oozing. After the injury there is usually a long interval, of several days at least, before the hemorrhage is large enough to cause symptoms of intracranial pressure. Then headache appears, severe and persistent, sometimes over the seat of the hemorrhage. This headache lasts a week or two and is gradually followed by slight mental changes, such as absent-mindedness, sleepiness, forgetfulness, etc. This "prodromal period" generally lasts about six weeks. Then, rather suddenly, the symptoms become much worse and the drowsiness quickly deepens to coma. This may be ushered in by violent headache and vomiting. The coma is of a remarkable type, undergoing curious spontaneous

variations, so that the patient passes from consciousness to unconsciousness, and vice versa. There is no other clinical condition in which this feature is so striking. The patient in the intervals appears mentally dull, but not confused. Responses to simple questions are made very slowly, but quite intelligently, so far as they go. The physical signs of focal cerebral lesion are variable, slight, and elusive. Pressure by the effused blood on the motor cortex may produce monoparesis or hemiparesis of leg and arm, the face generally being unaffected. An extensor plantar reflex and diminution or loss of the abdominal reflex are perhaps the most valuable unilateral signs. The extensor response may, however, be bilateral, even with a unilateral lesion, probably from dislocation of the opposite motor cortex against the cranium. Pressure on the midbrain may produce ocular or pupillary changes. Optic neuritis may develop. In unilateral lesions it is usually most marked on the side of the lesion. In bilateral lesions it tends to be most intense on the side of the greater lesion. Respiration is often peculiar—like that of a healthy person sleeping soundly—but a little exaggerated in amplitude and with expiration slightly emphasized. This can be observed when the patient is apparently awake. The hemorrhage is often bilateral, thereby confusing the clinical picture. Correct diagnosis is of great importance, since the condition is amenable to surgical treatment, and if the blood-cyst be evacuated and drained, complete recovery may occur. Spontaneous recovery is unlikely. Chronic subdural hemorrhage is probably identical with the condition known as *pachymeningitis haemorrhagica interna*, which is associated with general paralysis, with chronic alcoholism, and with senile dementia. The condition has usually been assumed to be a spontaneous subdural venous hemorrhage, but, as Trotter points out, alcoholic and insane patients are exactly those who are particularly exposed to the moderate kind of injury which is especially prone to produce chronic subdural hemorrhage, and the patient is not likely to remember or make much of the accident."

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CLINIC OF DR SIDNEY STRAUSS

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DISEASES OF THE BLOOD WITH SPECIAL REFERENCE TO HEMORRHAGES

Classification—There is still considerable discussion in the basic sciences as to the dual or the common origin of all blood-cells. There is, however, very little doubt that in the adult as distinguished from the embryo the small lymphocytes multiply for the greater part in the lymphatic tissues, and that all other blood elements arise largely in the active bone-marrow. If this is true, and clinical medicine seems to bear it out, then all true blood diseases are system diseases, that is, diseases of either the blood-forming organs, principally bone-marrow (myelogenous), but also the spleen and endothelial tissue elsewhere or of the lymphatics, according to Naegeli.

As diseases of the lymphatic system we have

Lymphatic leukemia, acute and chronic

Pseudoleukemia, or aleukemic lymphadenosis

Hodgkin's disease, or malignant granuloma

It is questionable whether the last should be classified as a true blood disease, but it is usually included. We shall discuss this later.

Diseases of the blood-forming organs, bone-marrow, spleen, endothelium, may include hyper-, hypo-, or dysfunction of any of the numerous elements of the bone-marrow. Thus we may have (1) pernicious anemia, a hypo- or dysfunction of that part of the marrow which forms the red cells, (2) polycythemia vera—a hyperfunction of the same elements, (3) myelogenous leukemia—acute or chronic—a hyperfunction of that part of the marrow which goes to form the whites, and (4) purpura

hemorrhagica (case to be taken up later), in which there is complete absence of whites together with a low blood-platelet count.

These represent most of the true blood diseases. The cause of these disturbances of function of the bone-marrow or of the lymphatics is not known, and in some instances which we shall touch upon later it is questioned that these are true system diseases. It is contended that they are diseases of the spleen or, as in pernicious anemia, are due to blood destruction primarily.

As an example of diseases of the lymphatic system the following case of Hodgkin's disease or malignant granuloma is presented.

Case I—A male, aged thirty-nine, was admitted to the Cook County Hospital on October 30th and died December 10th. On admission the patient complained of tingling and numbness of the hands and feet of twelve years' duration, itching of the skin, with eruption, generalized glandular swelling, swelling of the penis and scrotum, and cough.

The swelling began first in the neck, causing moderate difficulty in respiration, together with pressure and congestion in the neck. The mass slowly decreased in size. Contrarily, the mass in the inguinal region increased much in size.

The only significant fact in the past history was a chancre twelve years before.

Examination showed a generalized maculopapular eruption, excoriations, scalp formation and some secondary infections. The sequence of events was urticaria, scratching, infection, scarring. *There was almost total absence of hair.* The cervical, axillary, and inguinal glands were enlarged, firm and free. There was increased mediastinal dulness. Irregular nodular masses were palpable throughout the abdomen. The penis was swollen and edematous. The scrotum was the size of a grapefruit. The patient became gradually weaker and more dyspneic up to death.

Laboratory Findings—Urine was negative. Blood showed 80 per cent hemoglobin, 4,600,000 red cells, and 6800 white cells with a normal differential count.

This is, on the whole, a quite characteristic case of Hodgkin's disease with most marked involvement of the inguinal glands and consequent pressure therefrom. The itching is often observed in this and other blood diseases. There is little in this case to make us think we are dealing with a disease of the blood. Usually, however, there is a change in the blood-picture, and Bunting claims that he can diagnose Hodgkin's from the blood. In the beginning there is an increase, relative or absolute, of the large mononuclears. Later there may be a marked leukocytosis with polymorphonuclear neutrophils predominating. I have seen cases with counts of 30,000 or 40,000. There may also be an increase in the eosinophils. From this blood-picture it would seem that there was disturbance of the bone-marrow or that the lymphatic system had something to do with the genesis of the whites—if we consider Hodgkin's a blood disease. The picture in a leukemic lymphadenosis is quite different. There we have a relative increase in the small lymphocytes. This disease appears, however, to be a true proliferation of the lymphatic tissue, whereas in Hodgkin's we have a destruction and replacement of the lymphatic tissue by granulomatous tissue. May not the increase in cells from the bone-marrow be an attempt at compensation? The total loss of hair in this case is, so far as I can find, unique, and I have no explanation for it.

Without further discussion of diseases of the lymphatic system, we shall take up some of the diseases of the bone-marrow. Common to most of them is hemorrhage in one form or another. Consequently, I think it appropriate to study first a disease which may or may not be classified as a blood disease—*purpura haemorrhagica*. The case under discussion is, I think, distinctly, either primarily or secondarily, a disease of the bone-marrow. In no other way can the findings be explained. The case in short is as follows:

Case II.—A male, aged twenty-two years, entered the Cook County Hospital on April 8th and died April 10th. His complaints on entrance were bleeding from the nose, weakness, and discolored areas over the entire body, arms and legs, of four days'

duration. The disease began with a sore throat and headache, quickly followed by nosebleed and spots on the body. Nosebleed continued and was present on entrance.

Examination at this time revealed a young man, markedly pale, covered with purpuric and ecchymotic spots over the entire body, including the mucous membranes, varying in size from a pinpoint to 3 or 4 inches in diameter. There was a systolic murmur at the apex. The spleen was not palpable. The patient continued to bleed despite horse-serum and coagulose, and died in coma two days after admission.

His temperature on entrance was 104° F., pulse 124, and respiration 28, and they continued the same until death.

The urine showed large amounts of albumin, many red cells, and a few lymphocytes. The blood on April 8th showed hemoglobin 37 per cent, 2,690,000 red cells, and 250 white cells. In the stained smear only 3 small lymphocytes were found. The platelet count was 25,000. Coagulation time was twelve minutes. On the 9th the red blood-cells had gone down to 1,840,000.

The main findings at postmortem were extensive petechial and ecchymotic subcutaneous, submucous, and subserous hemorrhages, generalized anemia, melena, hemorrhage into the left kidney pelvis, hematuria, *hypoplasia of the bone-marrow*, perityphlitis following appendectomy, fibrous pleuritis. The blood and bone-marrow at postmortem showed absence of leukocytes. The spleen was not enlarged. *Streptococcus hemolyticus* was found in a culture from the heart's blood.

This case is characterized chiefly by extreme hemorrhages. It was ushered in by a sore throat, followed by a continuous high temperature. In spite of this there was an extremely low white count, in fact, a complete absence of all forms of leukocytes which come from the marrow. The cause appears to have been an infection, probably *Streptococcus hemolyticus*, gaining entrance through the throat and doing its greatest damage to the bone-marrow, destroying or inhibiting the myeloid tissue and the megakaryocytes, with consequent hemorrhage. The question is whether we are dealing with a case of *purpura haemorrhagica* so-called or with an aplastic anemia.

Acute leukemias may often begin with sore throat (marked necrosis of tonsils, alveoli, etc.) and are characterized by profuse hemorrhages, but in our case we have almost an absence of whites rather than an increase. It is this apparent contradiction in diseases of the blood, namely, that we may have hemorrhages in leukemias as well as in cases with extremely low white counts, in anemias as well as in polycythemias, that made me think it would be worth while taking up this particular feature for discussion.

First, it will be well to review the problem of hemorrhage and coagulation in its general aspects. Howell's¹ theory of coagulation is the one ordinarily accepted in this country. According to him there are five factors concerned—prothrombin, antithrombin, thromboplastin, fibrinogen, and calcium. All except thromboplastin are present in the circulating blood. Prothrombin is held in combination with antithrombin, thus preventing intravascular clotting. When however, there is bleeding "the antithrombin is neutralized by the thromboplastic substance of the tissue juices (blood-cells, endothelial cells, and platelets). The liberated prothrombin is activated by the calcium, and the thrombin which results converts soluble fibrinogen into insoluble fibrin or the clot." The normal clot quickly retracts and expresses serum. This is said to be due to the presence of blood-platelets. We have, then, to observe in any hemorrhagic disease (1) the coagulation time, (2) the bleeding time, dependent upon the potency of the tissue juices, the mechanical and chemical action of the blood-platelets and the elasticity of the skin, (3) the retraction of the clot and enumeration of the blood-platelets, (4) the quantity of prothrombin and fibrinogen. Detection of antithrombin is too complicated for clinical use. There is no evidence at present, according to Hurwitz, that any form of hemorrhage is referable to an abnormality of calcium in the blood. It has been shown that the bleeding in itcerus is due to the binding of calcium by the bile-pigments and not to a diminution in this element. Finally, bleeding may be due to a fibrinolysis, or dissolution of the formed clot by some abnormal ferment.

¹ Howell quoted by Hurwitz Amer Jour Med Sci., 154, 639

With these facts before us, we shall take up the cause of hemorrhage in each case. In the case before us we have as the cause of hemorrhage the marked diminution of the blood-platelets—25,000 instead of 150,000 to 250,000. With this should go an increased bleeding time and non-contractility of the clot. If hemorrhage is due to diminution of platelets alone there is no increase in the coagulation time. This form of bleeding is characteristic of purpura haemorrhagica. In aplastic anemia there is not only a diminution in platelets but also in prothrombin, with consequent increase in both bleeding and coagulation time. In our case there is an apparent slight increase in coagulation time which, however, has not been checked, nor was the bleeding time taken. We must, therefore, resort to other methods in making a differential diagnosis. The picture here is one of profuse hemorrhages, in contrast to the anemia which is not so marked. On the other hand, we have a complete absence of all myeloid elements, an aplasia of the bone-marrow. Again the disease is acute, the whole course being six to eight days, and is undoubtedly due to some infection. The logical interpretation is that there was an infection which caused a hypoplasia of the bone-marrow and consequent hemorrhages. Should we call such a disease an acute purpura haemorrhagica or an acute aplastic anemia due to sepsis? As Minot suggests, both diseases have much in common. On account of the preponderance of hemorrhages, the acuity of the disease, the only slight increase in the coagulation time, we have called this a purpura haemorrhagica with hypoplasia of the bone-marrow, we might have called it aplastic anemia with symptomatic purpura haemorrhagica in the absence of tests for prothrombin.

Having discussed aplastic anemia, it is appropriate to take up pernicious anemia.

Case III—The patient, a male, fifty-seven years of age, admitted to the Cook County Hospital on October 7th, died on November 3d. There was no postmortem. His only complaint was dyspnea, beginning ten days before and confining him to bed. He had a marked emphysema, and the diagnosis on

entrance was cardiac decompensation secondary to this His pallor and yellowish tint were, however, striking and blood examination showed hemoglobin of 40 to 45 per cent, 1,400,000 red cells and 3500 white cells, with a differential count of 43 per cent small and large lymphocytes, 6 per cent. eosinophils, and 49 per cent polymorphonuclears The reds showed marked polychromatosis, poikilocytosis, and anisocytosis, many megalocytes and microcytes On this the diagnosis of pernicious anemia was made The tongue was atrophied There was a systolic murmur (acute endocarditis was thought of as there was a slight fever from the beginning) Numerous hemorrhages were found in both eyes Toward the end there was noticeable bleeding from the gums The spleen was at no time palpable Just before death in coma the hemoglobin was 15 per cent and the reds 260,000 The blood was extremely thin and serous The smear showed poikilocytes, megalocytes, and megaloblasts The stomach contents showed no free acid and a total of 12

To be noted here are the absence of remissions, the apparent acuteness of the illness, a course of about six weeks, the absence of splenic enlargement There were no noticeable cord changes The blood-picture was, however, quite characteristic The hemorrhages in the retina are also typical The blood-picture in this disease is due to blood destruction and to abnormal blood formation Naegeli thinks it is due to disease of the blood-forming organs; principally the bone-marrow Others think it is primarily a hemolytic anemia There is no increased fragility of the reds, destruction of the reds is, however, evidenced by positive tests for bile-pigment in the blood by the indirect method of van den Bergh or the delayed reaction of Fouchet. That all elements are involved is shown by the low white blood-count, and at times the appearance of myelocytes and the diminution in blood-platelets The hemorrhages in this disease are probably due to the diminution in blood-platelets and at times to the diminution in prothrombin Consequently, there may be both an increased coagulation and bleeding time The etiology is unknown—stereotype for most diseases of the blood

We have already had a case in which there was diminution in the white cells. Next in order is one with a marked increase.

Case IV is that of a negro male, aged thirty-nine, admitted to a surgical ward of the Cook County Hospital on January 8th as a cellulitis of the thigh. His complaints were swelling and pain of the right thigh of seven days' duration, with sudden onset. Examination showed a large tender swelling on the anterior surface of the thigh which was soft and fluctuating. The surface was hot and shiny. The spleen was palpable 4 inches below the umbilicus, the liver three fingers below the costal arch. On the following day aspiration of the thigh gave bloody fluid.

Additional history revealed that the patient had had epistaxis for three months, asthma for four or five months, and dyspnea for the last six months. He had also noticed profuse bleeding from slight cuts and bleeding from a larger cut for one hour, during the last two years.

The blood-picture was as follows: 512,000 white cells, 50 per cent myelocytes, 44 per cent polymorphonuclears, 2 per cent myeloblasts, 2 per cent eosinophilic myelocytes, 2 per cent basophils, hemoglobin, 55 per cent, red cells, 2,800,000, 2 megaloblasts.

On January 17th the white count was 752,000, differential practically the same as above, polychromatophilia and anisocytosis were reported, a picture of marked hyperactivity of the bone-marrow, especially the myeloblastic tissue, but also of the erythroblastic. He was then transferred to the medical ward, with the diagnosis of splenomyelogenous leukemia and hematoma of the thigh.

At this time a swelling of much the same character as that over the thigh was noted over the left scapula and in the folds of the left axilla. There were a few enlarged glands of the neck. Aspiration of thigh again revealed pure blood. A gland was removed from the groin with rather severe hemorrhage. Section showed leukemic infiltration of a lymph-node, consisting mostly of large mononuclears. On February 11th a chronic suppurating otitis media was noted. Under treatment with benzol and x-ray.

and then x-ray alone, the white blood-count went down to 102,000, and the spleen extended to only 5 cm above the umbilicus. The hemoglobin, however, remained at 3½ per cent and reds at 2,210,000. The swelling in the axilla disappeared rapidly, and that in the thigh finally cleaned up. When he got up a right foot-drop was noted, probably due to pressure from the hemorrhage or clot in the thigh.

The diagnosis here is obvious. The interesting points of this case are the important part which hemorrhages played in his invalidism, and the location of the hemorrhages. Unfortunately, no work was done to arrive at the cause of the hemorrhages. However, from the history and from the fact that the patient bled profusely at the removal of a gland, we might conclude that either the bleeding time or the coagulation time, or both, were increased. With the increased formation of all bone-marrow elements it is natural to assume that the blood-platelets as well would be increased. They are increased or normal in myelogenous leukemia unless the megakaryocytes are crowded out by the myeloblastic tissue. We must then look for some other cause of the bleeding in myelogenous leukemia. For lack of other causes it has been thought that the bleeding is due to fibrinolysis from increase in the blood ferments and also to an increase in the antithrombin. It is also suggested that some of the hemorrhages may be due to the infiltration of the vessel walls by the myeloid cells. This may have been the cause of the massive hemorrhages into the loose areolar tissue of the thigh and axilla. Whereas hemorrhages usually play the most important rôle in acute leukemias, in chronic leukemias they are not often in evidence. In lymphatic leukemia there is an increased coagulation time and a diminution in the platelets, supposedly due to the partial crowding out of the megakaryocytes by lymphatic tissue.

Our final cases also deal with an increased activity in the blood-forming organs.

Case V—This case is that of a male, sixty years old, admitted to the Michael Reese Hospital on August 14th, complaining of

fulness in the abdomen of one year's duration, abdominal pain and distended abdomen for four months, constipation, and loss of weight. The pain is in the left upper quadrant. He had typhoid four years previously (possibly typhus). He walked into the ward and has at no time been bedridden.

Examination on admission showed a generalized adenopathy, marked cyanosis or pinkish color of the cheeks with dilated venules; deep purplish color of the tongue and the mucous membranes of the mouth. Tongue is smooth and atrophied. The abdomen was distended, the left part being most prominent. On palpation the spleen is found to fill about three-fourths of the abdomen extending from the sixth rib in the left axilla to the symphysis pubis and well beyond the median line. The liver edge is palpable three fingers below the costal margin, smooth and firm. The arteries are palpable and tortuous. Purpuric spots, varying in size from a pinpoint to 5 cm in diameter, were noted on the dorsum of the leg. The blood-picture was as follows on repeated examination: Hemoglobin 90 to 120+, red cells 7,010,000 to 9,760,000, white cells 33,950 to 51,300, differential count, polymorphonucleus 77 to 85 per cent. Differential made by Dr Howell showed polymorphonuclears 85 per cent, 4 large mononuclears, 4 small mononuclears, 1 eosinophil, 4 basophils, 2 myelocytes, 2 megaloblasts. In the beginning many nucleated reds were seen, as well as megakaryocytes and an occasional myelocyte.

There was a large amount of albumin in the urine, many red blood-cells and white blood-cells, and an occasional hyaline and granular cast. The blood-pressure was 140/90 and 128/90. The blood nitrogen was slightly increased, urea nitrogen 24, non-protein nitrogen 64. The fragility test showed partial hemolysis at 44, complete at 34—somewhat high for the complete. Wassermann test was negative.

After a few α -ray treatments at the hospital he was sent to the dispensary, where treatments have been continued and where he is still under observation.

Soon after leaving the hospital he developed a marked hematuria, which ceased in time, though there is still a large amount of

albumin in the urine The spleen during the first few months diminished in size, reaching only the umbilicus and two fingers to the left of the median line, where it has remained for the last year There have been no external hemorrhages for the past year The blood on August 14, 1924 showed 90 per cent hemoglobin, 6,960,000 red cells, 15,000 white cells, and 85 per cent polymorphonuclears

There is no doubt that we are dealing here with a hyperfunction of the blood-forming organs, principally of the reds, in other words, a polycythemia vera The remarkable points are the *extreme* enlargement of the spleen and the high white count The polymorphonuclear neutrophils, however, are overwhelmingly predominant in contrast to the previous case In contrast to that case, too, we have a plethora of reds and hemoglobin instead of an anemia Cases of myelogenous leukemia have been reported with a high red count, and with the extremely large spleen one might wish to consider this such a case All the findings are consistent with a Vaquez disease and not with a myelogenous leukemia Both diseases have much in common, and cases have been reported which at the start gave every appearance of being a polycythemia, but which ended with a typical picture of a leukemia with an anemia Polycythemia represents a hyper- or a perverted function of the erythroblastic tissue and myelogenous leukemia of the myeloblastic, both normally functioning in the bone-marrow

The discussion about polycythemia vera is mainly as to the seat and cause of the disease—is it a disease of the bone-marrow or of the spleen? The enlarged spleen is a constant finding and may reach an enormous size, as in our case If, however, it were a primary disease of the spleen, extirpation should benefit the patient, as in Banti's disease, but the contrary is true patients get worse or die from extirpation It is supposed that the large spleen is due to the destruction or storing of the reds in this organ and that the disease is due to hyperactivity of the bone-marrow from some unknown cause, such as a diminution of the O₂ carrying power of the hemoglobin, which is not borne out, or a diminished destruction of reds, against which is the fact that urobilin is found

in the blood and bilirubin in increased amount in the urine and feces at times. Here again we have our stereotype—etiology unknown.

As in our preceding cases hemorrhages are in evidence in the skin and kidneys in this case. The cause of hemorrhage is thought to be due to abnormal filling of the vessels. The blood volume is increased due principally to the increase in reds—80 per cent being corpuscles and 20 per cent plasma. The blood flow is retarded, due to the thickening of the blood and increased viscosity, all of which causes distention of the vessels, which can be seen early in the retina, and later in the distended peripheral vessels. These conditions lead as well to the formation of small thrombi. The increased viscosity also leads to an increased coagulation time and a clot that may not retract in spite of a normal number of platelets.

Another type of polycythemia has been described by Geisbuck, the so-called hypertonic type often appearing in elderly individuals. The following case seems to be one of this sort.

Case VI.—A male, aged forty-five, was first seen at the Michael Reese Dispensary in July, 1912. He then complained of pain in the right iliac region, severe, cramp-like in character, coming on at his work when he pressed shoes against his side. Examination revealed marked redness of the sclera, redness and marked dilation of the facial vessels, tenderness over the gall-bladder and liver, palpable and tortuous arteries, some albumin and a few casts in the urine, and blood-pressure of 140 systolic. He was referred to the hospital, where an enlarged spleen about two fingers beyond the costal margin was found, as well as cyanosis and a high red count. The history at this entrance is not at hand.

He continued to suffer from headache and dizziness, and was again referred to the hospital on July 2, 1913. His complaints at that time were as given above. The findings were as follows: Injection of conjunctiva and sclera, dilation of facial vessels, redness of nose and ears, some cardiac enlargement, spleen 3 cm below costal margin, red blood-cells 7,800,000 to 9,120,000,

hemoglobin 109 to 120, white blood-cells 7200 to 11,000, polymorphonuclears 78, small mononuclears 14, large mononuclears 8, blood-pressure 142 Urine showed no albumin

-He left the hospital improved, but returned to the dispensary in January, 1914 complaining of marked dizziness Findings as above, in addition albumin +++, red blood-cells 5,524,000

The next year (1915) he again returned, complaining of fainting spells He was markedly cyanosed and the heart had increased in size Blood-pressure was 146 He continued about the same, sometimes better, sometimes worse, up to 1920, when he returned with a right hemiplegia of sudden onset At this time his liver was palpable at the umbilicus, not tender, his heart was as above Blood-pressure was 178/118, albumin++

He was last seen on December 10, 1921, with the liver three fingers below costal margin and spleen two fingers Heart 12 cm to the left of midsternal line, pulse 96, blood-pressure 148/122, no edema Red count was not made

It may be questioned whether we are dealing here with a true or a secondary polycythemia It seems to me to be typical of the hypertonic type of polycythemia In the beginning there was no decompensation, yet the reds were as high as 9,000,000 The whites, however, were not much increased, and it does not give the picture of hyperactivity of the bone-marrow that the preceding case did We have the enlarged spleen and the slight increase in blood-pressure that are characteristic of the hypertonic type of polycythemia The course of the disease with hemiplegia is also compatible with this disease The arteriosclerosis, cardiac enlargement, and albumin may indicate a nephritis, but this would not explain the erythema Fitz says "It would seem reasonable to agree with Orlowski and believe that this condition represents a combination of erythema and arteriosclerosis with hypertension" The hemiplegia common in this disease may be due to the polycythemia or to the arteriosclerosis

The 6 cases here presented cover in a way the field of blood pathology They emphasize that much is still to be learned regarding the etiology and, consequently, the classification of

diseases of the blood. Special emphasis has been laid upon hemorrhages in diseases of the blood with the view of bringing out the diversity of the causes of hemorrhage, and the great opportunity which the study of diseased individuals offer in arriving at the solution of these causes.

CLINIC OF DRs JULIUS H HESS
AND PHILIP ROSENBLUM

SARAH MORRIS CHILDREN'S HOSPITAL OF THE MICHAEL REESE
HOSPITAL

GASTRO-INTESTINAL HEMORRHAGE IN CHILDREN

RECENTLY several interesting cases of gastro-intestinal bleeding have come under our care. It so often happens that the etiology is hard to ascertain. We do not refer to the cases of melena neonatorum which come under another category. In the 4 cases to be presented the etiology was ascertained at operation or by x-ray.

CASE I DUODENAL ULCER IN A GIRL EIGHT AND A HALF YEARS
OF AGE

Birth History—Normal full term baby. Breast fed to four and a half months. Later albumin milk and cow's milk. Development normal.

Present Complaint—Child was apparently well until January, 1925, when she began to have slight cramp-like pains in the epigastrium. These came in short intervals and passed away. They were unassociated with vomiting, belching or diarrhea. Cramp-like pains continued for forty-eight hours, when, on January 21, 1925, the child began to expel a very foul flatus. The mother remarked it had the odor of old blood. This continued for two days. On January 24th the child was given 6 ounces of citrate of magnesia, but this did not relieve passage of foul gas. The afternoon of same day child fainted at a dramatic art recital. That evening one of us was called. Patient was quite pale, pulse rapid. Black, thin stool was passed. No vomiting. Temperature 100 4° F by mouth.

Appetite had been fair. There never had been any history of pain in the abdomen in relation to food or otherwise, or of any previous acute or chronic abdominal condition. There was no constipation.

Past History—Eczema since four months old. Influenza when two years old. Pertussis at five years, measles when four years old, occasional cold and sore throat.

Family history negative.

Child was observed at home for four days. A slight elevation of temperature to 100° F occurred, with intermittent



Fig. 273.—Defective filling of bulbus duodeni: A constant finding

passage of dark stool. Finally, January 29th, condition seemed to get worse suddenly after passage of dark, copious, bloody stool, and patient was sent to Sarah Morris Hospital. Temperature on admission 102° F rectally. Pulse 128, respiration 28. There was no abdominal tenderness or rigidity. Hemoglobin was 40 per cent. Red blood-cells 1,730,000, white blood-cells 15,700, of which neutrophils 77, small mononuclears 18, large mononuclears 4, transitionals 1, showing an anisocytosis and poikilocytosis. Urine essentially negative. Proctoscopic ex-

amination with continued bowel washing revealed considerable dark blood. No point of hemorrhage was visible. Owing to patient's condition a blood transfusion was performed by Dr A A Strauss, 350 c.c. of blood from an unrelated donor was given. The temperature and pulse returned to normal after four days. Hemoglobin improved so that ten days after transfusion the hemoglobin was 65 per cent, red cells 3,500,000, and white cells 6200.

Child left the hospital two weeks after admission, apparently well, with the understanding she would return in two or three weeks for gastro-intestinal γ -ray which was done. The report of Dr R A Arens, Roentgenologist, showed the probable cause for hemorrhage (Fig 273).

Fluoroscopic Stomach is orthotonic in type, extending down to the crest line. No defects. Gastric motility four and a half hours.

Duodenum The bulbus duodeni does not fill out well at any time even under atropin appearing constantly defective. In twenty-four hours the barium column has reached the rectum. No colon defects. The appendix can be visualized, but is not tender on palpation.

After forty-eight hours colon is empty.

Opaque enema The colon fills readily throughout. No defects noted. There is a slight tubulization of the descending and sigmoid loops.

Roentgenographic The films confirm the fluoroscopic findings, disclosing a typical ulcer defect of the bulbus duodeni.

Proctoscopic examination was repeated, but was again negative. Following admission to hospital the child was placed on strict ulcer management, which was continued at home. To date (February, 1926) there has been no recurrence of hemorrhage. There has been a steady increase in weight. Hemoglobin is 85 per cent. There is no abdominal discomfort of any kind. In fact, the mother states child is in better health than ever before. Gastro-intestinal γ -rays will be repeated to see if any change has occurred in the ulcer.

Discussion—Most of gastric or duodenal ulcers reported in children have occurred in infants under two years. Gastric and duodenal ulcers in children do not have characteristic pain usually associated and diagnostic in the adult. Since a good many ulcers are healed under medical treatment, even some of the chronic indurated ones, surely medical treatment is indicated as the early method of treatment. In this particular case a cure seems to have taken place. The mother, however, was told that should the hemorrhage be repeated operation might become imperative.

CASE II CONGENITAL ATRESIA OF THE DUODENUM BY ADHESION

The second case is that of a child aged ten and a half months. He was first admitted to our service when thirteen weeks old, with a history of losing weight and vomiting of food. Vomiting of food first occurred when he was one week old, and was quite pronounced at end of puerperium. When the child was two weeks old the mother had gall-bladder trouble and lost her milk. The child was transferred to the Children's Hospital because of vomiting. It was placed on chymogen milk and atropin sulphate, grain 1/1000, four times a day, with the result that it gained on an average of 1½ to 2 ounces a day, child left the hospital August 21, 1924, improved. It was then five weeks old.

The child next came under observation when ten months old because of fever, cough, and vomiting. After leaving the hospital in August, 1924 there was practically no vomiting for a few days. Then the child began to vomit on an average of once a day. This has been increasing during the past month. The cough began one week before admission. Fever has been present since onset of the cough. Bowels move daily without cathartics or enemas. He has lost 1½ pounds in a week.

Physical Examination—At this time the infant appeared extremely anemic and severely ill. Temperature was 103° F on admission. Temperature persisted for five days. There were a few rales in both lungs, but no consolidation. There was visible peristalsis of stomach and marked evidence of



Fig 274—Dilated pyloric ring and duodenum with atresia of duodenum



Fig 275—Postoperative result
tomogram Barium passing through gastro-enterostomy
Pylorus closed

dehydration. The white blood-count at this time was 44,000, with neutrophils 85, small mononuclears 8, mononuclears 7, hemoglobin 45 per cent. The Von Pirquet test was negative. Urine was negative. Stool examination at this time showed microscopic and chemical blood. Stomach washings returned dark brown particles. A blood transfusion of 100 cc was given intravenously and 100 cc of normal saline sub-



Fig 276—Same as Fig 275

cutaneously. The saline was repeated daily for four or five days. The child was given breast milk and made a steady improvement, so that by March 13th an x-ray (Figs 274, 275) was taken of the gastro-intestinal tract. The stomach showed 95 per cent retention after four hours. The stools became normal in color and the baby was making a regular daily gain when discharged March 9, 1925.

March 12, 1925, four days after second discharge from the hospital, the child returned because of vomiting which started forty-eight hours before admission, and was becoming progressively worse. The vomitus was tinged with blood. At midnight before entrance the child vomited large quantities of brownish material, and he became almost prostrated. He had



Fig 277.—Stomach emptying rapidly. Gastro-enterostomy patent. Pylorus closed. Much of barium in jejunum and ileum.

four or five watery dark brown stools daily for two days before entrance

On account of the critical condition and constant bleeding a transfusion of 120 cc of whole blood was given and a laparotomy performed by Dr A. A. Strauss

Upon opening the abdomen what appeared to be a very large stomach was projected into the wound. Upon closer ex-

amination it was noted that the pyloric muscle consisted of a thin band of muscle-fibers encircling the organ in its lower third. The pylorus itself was patent and 12 cm in diameter. The duodenum was dilated to the size of approximately 8 cm in diameter and appeared as if part of the stomach. About 3 inches below the pyloric ring there was a marked constriction of the duodenum due to an encircling band of adhesions binding it to the posterior abdominal wall beneath the liver. Because of



Fig 278.—Four hour plate Stomach almost emptied

its firm attachment and the difficult position attempts at releasing it were abandoned. A gastro-enterostomy with pyloric closure was performed.

The patient made an uneventful recovery. α -Rays taken March 30, 1925, or about twenty days after operation, disclosed a gastro-enterostomy that practically drains the stomach completely in four hours. The stools were free from blood for several days before final discharge, April 4, 1925.

This child has continued to gain and has been free from vomiting and blood

Discussion — This case began like an ordinary pylorospasm, and was relieved by atropin. Vomiting recurred, also hemorrhage. The adhesions to the posterior abdominal wall beneath the liver produced a stricture of the duodenum. Hemorrhage was in all probability due to venous engorgement in the dilated duodenum. Finally, uneventful recovery after transfusion and gastro-enterostomy.

CASE III BLEEDING MECKEL'S DIVERTICULUM

The third case admitted to the service of Dr I A Abt is that of a baby eleven months of age, full term, weight 24 pounds. He was breast fed, supplemented by cereals and vegetables.

Present Complaint — Hemorrhage from the rectum, fainting spells, fever and vomiting, loss in weight

Mother stated that bleeding from the bowel was first noticed about two months ago. Child had been sleeping when mother picked him up and noticed that his diapers were marked through and through with bright red blood. The next morning he bled again. That afternoon she noticed a small amount of blood in the diaper. The child felt fine during the entire time, sleeping and eating well.

The second attack came on a week before admission, September 4th, when the mother found a small amount of dark red blood in the diaper. The next night the child began crying and straining, trying to make the bowels move. He turned very pale and seemed to faint. The following day there was a small amount of blood in each bowel movement (two to three that day). The bleeding continued for the next two days. No blood or diarrhea was present for four days previous to entrance. He vomited once the day before admission. Temperature varied between 99° and 101° F.

Physical examination disclosed a very pale well-nourished baby, showing some evidence of tenderness throughout the abdomen. There were no definite areas of involuntary rigidity or evidence of free fluid. Liver and spleen were palpable.

Blood examination showed 30 per cent hemoglobin, 2,400,000 red cells, 11,000 white cells, with a differential count of 47 neutrophils, 48 small mononuclears, 4 large mononuclears, and 1 transitional. Urine was negative as was the Von Pirquet test.

Operation was advised. Because of the child's condition transfusion of 150 c.c. of whole blood was given. An attempt at proctoscopic examination was made before operation, but because of the presence of so much black, tarry feces the rectal wall could not be observed.

Operation was performed by Dr. A. A. Strauss. There was a distinct mass, about $\frac{3}{4}$ inch in diameter, in the right fossa about 8 inches from the ileocecal valve. This proved to be a Meckel's diverticulum. The bowel for a distance of several inches showed bloody content, as though this might be the source of the hemorrhage. The diverticulum was amputated. The mucosa upon opening showed ulceration and evidence of recent bleeding. The child made an uneventful recovery following operation.

Discussion—Gastro-intestinal hemorrhages in infants under two years of age without apparent cause should direct one's attention to Meckel's diverticulum as a possible cause. Meckel's diverticulum is inserted in the midgut of the fetal intestine. In the early weeks of fetal development the midgut has an extra-abdominal position in the vitelline sac. This gradually recedes into the abdomen, separating the vitelline vesicle at the umbilicus. If it is complete, there is a total separation of vitelline vesicle and midgut. Occasionally malformations occur due to vitelline duct remains. These may be located in the umbilicus, ileum, or appear as various bands in the peritoneal cavity. Usually they result from the failure of more or less of its structure to disappear completely.

Operation is essential to recovery in cases of bleeding Meckel's diverticulum.

This condition probably occurs more frequently than is diagnosed.

CASE IV SPLENIC ANEMIA

The patient, F. S., female, aged fourteen months, first entered the hospital on our service September 9, 1923. She was a full term child, delivered spontaneously, and weighing at

birth 7½ pounds There was no asphyxia cyanosis, or hemorrhage She was breast fed for eleven months Orange juice was added at the fourth month, cereals and vegetables at the eleventh month She weighed 19 pounds when one year old The complaint at the time of admission was hematemesis and hemorrhage from rectum

Two or three days before admission the child was not as playful as usual, refused nearly all feedings, and appeared pale On Saturday, September 7th, she was drowsy nearly all day At 11 A M the day before admission she vomited blood apparently without any cause This was bright red and black clotted blood, about 6 ounces in all At 6 P M the mother noticed bleeding from the rectum The succeeding stools were also tarry The child complained of thirst all day There were never any similar attacks or gastro-intestinal disturbances

Physical examination on admission showed a fairly well-nourished baby, quite anemic The tongue was dry and coated The pulse was very rapid, 200 per minute, and regular Heart was not enlarged Abdomen showed no rigidity Liver was not palpable, spleen just palpable Digital examination of the rectum was negative The child was passing black, tarry stools Temperature varied from 100 4° F (rectal) on admission to 104° F, returning to normal after blood transfusion, except for a secondary rise on September 15th due to a pyelitis

Blood examination on admission showed 30 per cent hemoglobin, 2,600,000 red cells, 13,000 white cells Differential count showed 60 neutrophils, 34 small mononuclears, 4 large mononuclears, 2 unclassified, coagulation time three and one-half minutes Von Pirquet test was negative

Blood transfusion was given on the second day after admission No fresh hematemesis occurred She continued to have tarry stools for ten days, following which she gradually improved, so that on discharge, October 5, 1923, the hemoglobin was 50 per cent, red blood-cells 3,500,000, white blood-cells 6800, and differential, 48 neutrophils, 39 polymorphonuclears, 10 small mononuclears, 2 large mononuclears, coagulation time two and one-half minutes

The child was discharged with a tentative diagnosis of splenic anemia in the early stage or bleeding gastric or duodenal ulcer

Six months later (April 26, 1924) the child returned to the hospital on the service of Dr I. A. Abt, with the complaint of not gaining properly, diarrhea, blood in the stools, and elevation of temperature

She was quite pale and drowsy. The spleen was large and firm, with a smooth edge. Temperature averaged about 100° F. Blood examination showed 65 per cent hemoglobin, 4,400,000 red cells, 5800 white cells, with a differential count of 40 neutrophils, 52 small mononuclears, 8 large mononuclears, coagulation time six and one-half minutes. Wassermann test was negative. A diagnosis of Banti's disease was made.

On May 23d, about four weeks after admission, the child vomited a considerable quantity of blood. Hemoglobin was reduced to 50 per cent, red blood-cells 3,500,000, and white cells 6800. The hemoglobin gradually dropped to 38 per cent, so transfusion and splenectomy were advised.

Roentgen examination at this time disclosed no evidence of ulcer of the stomach or duodenum. The stomach emptied in four hours.

On June 28th splenectomy was performed by Dr G. L. Davenport. The child steadily improved following operation. Pathologic examination of the spleen by Dr O. H. Schultz showed the following:

Diagnosis—Fibrotic splenomegaly (Banti's disease)

Gross—Spleen measures 10 5 x 7 x 4 cm. The capsule is smooth, tense, and free from adhesions. There are two shallow notches in the superior border and two in the inferior border. The tissue cuts with increased resistance. The cut surface is brownish red and shows numerous very fine fibrous trabeculae. The malpighian bodies are increased and prominent. The small amount of blood which escapes from the cut surface is pale, thin, and watery.

Microscopic—The spleen pulp is exceedingly fibrous and contains practically no blood elements. The trabeculae are slightly thickened and prominent. The malpighian bodies are

quite long and some of them are quite active. The centers of others consist almost entirely of reticulum cells. Occasionally there is a rounded mass of fibrous tissues which shows a few lymphocytes and gives the impression of being a fibrosed lymph-follicle. Because of the marked fibrosis of the pulp and the occasional appearance of the fibro-adeni, a probable diagnosis of Banti's spleen can be made.

The child had no more hemorrhages following removal of the spleen. On July 20, 1924 the hemoglobin was 58 per cent red cells 4,160,000, and white cells 9000. The child was seen again on August 22d for an acute respiratory infection. At this time the hemoglobin was 60 per cent, red cells 4,900,000, white cells, 14,000, with a differential count of 20 polymorphonuclears, 70 small mononuclears, 6 large mononuclears and 4 transitionals. Her general condition seemed much improved.

Discussion—Here we have a case of splenic anemia. The first symptom was hemorrhage in the gastro-intestinal tract, followed later by enlargement of the spleen and slight enlargement of the liver. The blood-picture was that of a chlorotic anemia, the hemoglobin being reduced more than the red blood-cells.

It was necessary in this case to differentiate between Gaucher's and Banti's disease. In the former there is often marked reduction in the hemoglobin without any appreciable decrease in the number of red cells. There is no ascites or icterus until severe hemorrhage sets in. There is, however, often a brownish pigmentation of the skin also thickening of the conjunctiva and pigmentation. Gaucher's disease frequently occurs in more than one member of a family and is of longer duration than Banti's disease.

In Gaucher's disease the microscopic examination of the spleen is characteristic. There are large irregular spaces appearing in the splenic tissue, which are filled with large cells, either round or oval. Their nuclei vary in size and shape. The normal pulp cells often entirely disappear. The bone-marrow contains these same large endothehal-like cells which are also present in the liver and in the lymph-nodes.

Other conditions which should be considered when dealing with hemorrhage associated with splenic enlargement are syph-

ilis, malaria, congenital hemolytic jaundice, Hanot's cirrhosis, leukemia, purpura, and Von Jaksch's pseudoleukemia

Syphilis can be excluded by a positive Wassermann and other evidences of lues

In malaria the finding of parasites in the blood and the temperature curves clinch the diagnosis. Splenic enlargements and hemorrhage due to intestinal parasites usually present an eosinophilia together with parasites or ova in the stools.

Congenital hemolytic jaundice shows an increased fragility of red blood-cells and an acholemic jaundice. This condition is quite rare in infants. When present there is evidence of acute blood destruction. It often occurs in more than one child in a family.

In Hanot's cirrhosis the liver is primarily involved, and the disease is associated with intense icterus, leukocytosis, and fever.

The leukemias are usually detected by careful blood examination. With the exception of the acute lymphatic leukemia they are quite rare. In the latter condition hemorrhages from all the mucous membranes may occur. The white count is greatly increased, elevation of temperature is noted, and the course is more rapid than in the other forms of leukemia.

Purpura haemorrhagica presents a marked reduction in blood-platelets—below 150,000—associated with petechiae, and usually a slight increase in the white blood-cells.

Von Jaksch's pseudoleukemia occurs in infants between the ages of six and twenty-four months. The white count ranges from 20,000 to 50,000, with an increase in mononuclears and the presence of myelocytes. The blood findings and spleen suggest the quite evident diagnosis.

A thorough general and blood examination is necessary in all cases of hemorrhage in order to institute proper therapy.

Banti's disease is accompanied by a marked enlargement of the spleen, in the later stages by enlargement of the liver, jaundice, and cirrhosis. There is a marked leukopenia, often to 1000. There is an increase in urobilin, which is the most characteristic sign of blood destruction. The eosinophils usually disappear and there is a marked tendency to hemorrhage.

CLINIC OF DR JACOB MEYER

MICHAEL REESE HOSPITAL

- I ORTHOSTATIC ALBUMINURIA
- II ACUTE GLOMERULAR NEPHRITIS
- III CHRONIC GLOMERULAR NEPHRITIS.
- IV ACUTE YELLOW ATROPHY

I ORTHOSTATIC ALBUMINURIA

THE patient is a young boy sixteen years of age who was first examined in 1920. At this time he had no complaint but in the course of physical examination albumin was found in the urine. The physical findings at this time showed a rather poorly nourished pale boy. The heart was normal in size. A systolic murmur was heard over the pulmonic area. The systolic blood-pressure was 110 mm., diastolic 80 mm. The spine showed no lordosis, but the posture of the boy was described as drooping. All other findings were negative except the albuminuria. Calcium oxalate crystals were observed in the sediment. The patient was advised to rest in bed, lying flat on his back for twenty-four hours. Examination of the morning specimen of urine for albumin gave a negative result. The boy was then told to run about the room, a fresh specimen of urine was collected, and albumin was now found to be positive. A diagnosis of orthostatic albuminuria was made. I wish to present this patient to you with his story for the past four years. He has had no intercurrent illnesses. As you see, albuminuria is still present and disappears from the urine after a night's rest. This chart (Fig. 279) is a record of the height, weight, and blood-pressure of the patient for the entire period. You notice that the patient has been underweight as compared to the average weight for

HEIGHT, WEIGHT, AND BLOOD-PRESSURE CHART

Date	Sept 20 1920	April 11 1921	Sept 2 1921	Dec 22 1921	May 1923	May 1925	Sept 1925	May 1924	Feb 1925
Age	11 5/12	11 11/12	12 4/12	13 2/12		13-10	14 4	14 9	16 9
Height	53 5 in	54 5	56	50 7	57	59 5/8	60 5	61 7	63 7
Weight	63 5	70	73	63 7	60	86 5	94	106	110
Average Weight for Height	70 4	75 7	79 1	61 8	63 8	95 1	97 2	104	117
Underweight Lbs Per cent	6 9	6 1			2 8		5 2		7
Overweight Lbs Per cent	9 8	7 7			3 5	7 1	5 5		5 9
Blood Pressure								2	
Systolic	110			74	112	118	112	116	
Diastolic	80				70	64		74	80

Fig 279.—Orthostatic albuminuria

his height I think this important, as it points to the relation of orthostatic albuminuria, to the changes in stature, body development, and growth of the young child as it proceeds from

puberty to adolescence. Very often this type of albuminuria is first observed at the onset of puberty and may persist for a number of years during active growth, only to disappear in later full development. The relation of orthostatic albuminuria to lordosis of the lumbar spine is well known and was first emphasized by Jehle. The blood-pressure readings may be considered normal for this boy. Patients with orthostatic albuminuria may present variations in the cardiovascular system such as a ptotic heart, as seen in the fluoroscope—accentuation of the second pulmonic and systolic functional murmurs. The blood-pressure is usually normal or low. What constitutes normal blood-pressure in a young child may probably be a subject of question, but the absence of any marked variation over a number of years in the presence of an albuminuria may be taken as evidence of absence of glomerular renal disturbance.

Of equal importance is the absence of cardiac hypertrophy. Whereas the presence of blood-pressure increase without cardiac hypertrophy may speak for a renal lesion, it is probably more correct to say that increase in blood-pressure, transitory or permanent if associated with cardiac hypertrophy and albuminuria speaks against a functional or orthostatic albuminuria. I wish therefore, to direct your attention to the importance of carefully examining the heart and watching the blood-pressure over a period of years. The diagnostic data of orthostatic albuminuria may be summarized as an albuminuria postural, occurring in young growing children, disappearing on rest, associated with normal or low blood-pressure and normal cardiac outline.

II ACUTE GLOMERULAR NEPHRITIS

The second patient is a young boy nineteen years of age. In February 1924 he was admitted to the hospital complaining of headache, vomiting, and swelling of the face. Three and a half weeks previous to admission he had chickenpox. He took the ordinary precautions, but about a week before admission he noticed swelling of his eyelids and complained of headache. This condition gradually increased in severity, and on the day of entrance he vomited, the headache was severe, and he noticed

that his urine was red. The previous history of the young man is not of interest. Physical examination showed a tall, young, pale boy with edema of face and eyelids. The heart was normal in size. Pulse was forceful and of high tension. Blood-pressure on admission was systolic 150, diastolic 70, temperature, 98.6° F., pulse, 60, respiration, 18, urine scanty, specific gravity, 1030, albumin strongly positive and many red blood-cells and casts, blood, hemoglobin 80, red cells 4,000,000, white cells 99,500, N P N, 94. A diagnosis of acute glomerular nephritis was made.

He remained in the hospital for a period of one month. With restriction of fluid intake and low protein salt-free diet the edema disappeared. The blood-pressure remained high for a period of one week and then dropped to 140/70. On February 18th he developed a sore throat, rhinitis and cough, and temperature gradually rose to 102° F. The urine, which had up to now contained only a few red cells, now showed an increased number and the blood-pressure rose to 140. X-Ray examination revealed a clouding of the left antrum. Fluids were now unrestricted. Elimination was now increased by magnesium sulphate and Dover's powder. The patient's condition improved daily. Temperature normal, fewer red blood-cells in the urine. Patient was then placed on an increased amount of protein 70 gm, fat 120 gm, carbohydrates 285 gm. Blood-pressure at time of discharge was 132/90. The patient was sent home and advised to remain on standard nephritic salt-poor diet.

One month later examination showed no change from previous condition on discharge. The patient was advised to continue the same routine. Three months later blood-pressure was taken, and reading was 125, no change in size of heart. Six months later the patient was seen again. This time the urine was free of any trace of albumin. In this period he has been on a fairly liberal diet. He eats meat twice weekly, eggs 1 to 3 daily. He has learned to avoid spices and condiments. As you see him, approximately eighteen months since onset, he is a well-nourished young man, the urine is now again negative. His blood-pressure ranges from 120 to 125 systolic and 70

diastolic There are no changes in the heart, no change in the lungs I present him as a case of acute glomerular nephritis, with complete healing I think it is safe to say this in view of the fact that he is free of any blood-pressure changes for a period of a year There is no evidence of renal disturbance His urine is of normal specific gravity and a water test shows that he is able to eliminate and concentrate

III CHRONIC GLOMERULAR NEPHRITIS

The third patient is also a young boy eighteen years of age He was first examined in 1921 The left ear was discharging pus and this had been present for a number of years The heart measured 8 5 cm to left of midsternal line and 2 5 cm to the right of midsternal line The blood-pressure was 120 mm systolic and 80 mm diastolic The urine was strongly positive for albumin and many hyaline and granular casts were also found Co-operation was not obtained from the family and the patient was not seen again for a time

In 1923, two years later he was seen with an acute tonsillitis The ear condition had healed The heart outlines were unchanged, the blood-pressure, however, was 148 systolic and 60 diastolic The urine now contained albumin, red blood-cells, hyaline and granular casts Patient was treated as an acute tonsillitis and acute nephritis Salicylates were not given The diet was restricted to fluids and fruit juices Hospitalization was not consented to The patient was not seen again until March 1924 In the interval he had had his tonsils removed The following changes were noted The left heart border was 10 5 cm to left of midsternal line The right 2 5 cm, the blood-pressure 140 systolic, 70 diastolic An acute bronchitis was also noted and the urine contained albumin, hyaline casts, no edema At the present time you see the patient is a robust, tall young boy He has no complaint The heart is 10 $\frac{1}{2}$ cm to left of midsternal line and 2 $\frac{1}{2}$ cm to the right The blood-pressure is 146 systolic diastolic 70 The urine is positive for albumin, and contains many hyaline casts There are no other physical findings of importance

I have presented this patient to you because he represents a record of a patient in the private practice of medicine, and will exemplify the difficulties you may encounter in the study and the control of certain patients. However, in spite of these difficulties let us analyze the story of our patient.

In the absence of any definite history we may be justified in assuming that this boy at one time had an acute infection, probably an acute otitis media, and that associated with it he had an acute nephritis which was not recognized. As he came to us in 1921 he presents a picture of either a (1) postnephritic albuminuria (2) healing with defect, or (3) a chronic glomerular nephritis, or (4) possibly a nephrosis.

If a complete healing of an acute nephritis, as exemplified by our second case does not occur, there remains varying types of clinical manifestation depending on the severity of the lesion. The first and the mildest of these is the so-called (1) post-nephritic albuminuria. No other symptoms or signs are present except the albuminuria, no changes in cardiovascular system, occasional red blood-cells, and the patient goes along through life without disturbance of any kind. A second form represents the so-called (2) healing with defect. The clinical findings are those of positive albumin, which increases with exertion and subsides with rest. Casts and a few red blood-cells are present. Kidney function is normal and there are no changes in the cardiovascular apparatus. The lesion, as a general rule, is not progressive. There may be a very slight tendency to edema. Mild infection may produce edema together with an increase in red cells. Such patients get along well on a moderately restricted diet. It is this type of patient which should be observed, but not frightened, as to his future.

The third possibility is the development of a chronic glomerular nephritis. Of greatest diagnostic significance is the gradual development of a high systolic blood-pressure. This may extend over a period of years, as in our case, but the increase is a constant one. In children it is important to make repeated examinations because, owing to psychic disturbance, vasomotor instability and many other factors variations of blood-pressure,

are so frequently observed. If a hypertrophy of the left ventricle is also demonstrated one may speak without doubt of the presence of a renal lesion. Such is the finding in our patient (and there is no valvular defect). At the present stage we may term this a "chronic glomerular nephritis." I do not think one need consider a true nephrosis in this case. Nephrosis as you know, is a degenerative disease of the kidney tubules. The etiology is much in question. The true nephroses are believed to be associated with constitutional disturbance, thyroid disturbances, lues (?) tuberculosis, chronic infections and recently it has been claimed that it is due to staphylococcal infections. The true nephroses are characterized clinically by marked edema, pallor, and weakness. The urine is of high specific gravity, albumin is present in as high as 5 to 10 per cent, hyaline fatty casts are present, and double refractile fatty substances are found in the sediment. The kidney functional studies of the blood are normal, but there is a difficulty in excreting chlorids. The nitrogen content of the urine is increased. The blood shows a loss of albumin—figures of 3 to 4 per cent against a normal of 6 to 8 per cent are often found. The blood cholesterol is increased. The blood-pressure is normal, the heart is normal in size. The patients may remain well for a long period. They may lose their edema. They may, but rarely ever pass into the stage of secondary contracted kidney. Death is generally due to a secondary infection—most commonly a pneumococcus peritonitis. The diagnosis of a true nephrosis is difficult at times and particularly in the edema-free stage. As distinct from nephritis, a nephrosis is characterized by the absence of red blood-cells in the urine, the absence of blood-pressure elevation and absence of cardiac changes. A chronic glomerular nephritis with edema may present the clinical picture of a nephrosis. Volhard speaks of this as a nephritis with a nephrotic "Einschlag." The differentiation may be difficult, but again, the presence of red blood-cells in the sediment, the increased blood-pressure, and the cardiac hypertrophy serve to differentiate. Double refractile lipoid bodies may be present in the urine in this type of case as well as in true nephrosis.

Let us now return to our patient. He has, as you see, a great quantity of albumin in the urine many hyaline casts. There are no red blood-cells, no double refractile bodies. The kidney function tests are normal. I believe the patient represents a form of chronic glomerular nephritis in an edema-free stage. The high blood-pressure and the heart hypertrophy point to the glomerular character of the renal lesion. Of course it is not without doubt that tubular changes are present. Let us now say a few words as to the probable course. As the young man advances in age signs of cardiac insufficiency will develop. They may become manifest by shortness of breath, cough, precordial distress, and, finally, a true picture of cardiac failure. Nocturia may be the earliest symptom of impaired renal function, but normal functional kidney tests may be obtained for many years. It is difficult to say how long such a case may go on. The development of repeated infections, cardiac symptoms, changes in the eye-grounds, and tendency to edema serve as a guide to the progressive character of the disease. The majority of these patients present themselves because of the edema and the cardiac discomfort, and the renal origin of the lesion may be entirely overlooked. Uremia may, but seldom does, occur in this type of case. It is more common in the secondary contracted kidneys, with signs of insufficient kidney.

Do not attempt rigid measures in the treatment of such a case. In the absence of edema and with normal kidney function all the usual restrictions should be moderated. Occupation should be restricted so as not to throw excessive work on the cardiovascular system. Dietary restrictions generally are made very severe, but, on the whole, I think this is unnecessary. It is sufficient to instruct the patient to omit spices and condiments. A low-protein diet is also advisable. With the development of edema, rest in bed, cardiac stimulation, and the simple measures of elimination should be advised.

IV ACUTE YELLOW ATROPHY

This disease is considered rare. I wish to present to you the history of a case which we recently observed, and then show you

the pathologic specimen. The patient, a woman forty years of age, entered the hospital July 1, 1925. She was apparently well up to three weeks before entrance, when she felt warm, perspired freely, but had no chill. Within a week there was jaundice, clay-colored stools, and a sensation of creeping under the skin. Following the onset of jaundice patient began to vomit once to twice daily. There was no association of pain at the time of vomiting, nor has there been any pain at any time in the entire course. The jaundice has become deeper. There has been no previous attacks of jaundice. The history is otherwise negative. The patient is married, has 4 children living and well, has had one miscarriage at three months, and one still-birth. There is nothing in the history or findings to suggest a recent pregnancy.

On physical examination, we observed an intensely jaundiced woman, who was drowsy. The icterus was most intense, so that the examining-room physician diagnosed obstructive jaundice. However, it was noted that although a high grade of jaundice was present, the liver was not palpable. The normal liver dulness was not marked. The abdomen was relaxed, there was no pain or local tenderness. The stools were clay colored, and on the first examination the Schmidt test for bile was negative. On the second and third examination a Schmidt test for bile was positive. The urine was definitely bile colored, contained a trace of albumin, but no leucin or tyrosin crystals were found on repeated examination. van den Bergh test was positive indirect and direct. The Wassermann was negative, the blood-culture negative.

The impression first held by most of us was that the patient had an obstructive jaundice. In the absence of a story of pain, or colic of any kind, the diagnosis of "obstructive jaundice" was questioned. I suggested that the patient might have a severe catarrhal jaundice which progresses into an acute yellow atrophy, or, as more commonly expressed, a case of acute yellow atrophy which begins with the symptoms of acute catarrhal jaundice. There was little in favor of such a diagnosis when the patient was first seen, except the story of progressive jaundice, repeated vomiting of bile without pain, the toxemia and the presence of bile in the stools, pointing to absence of complete

obstruction. The temperature at this time was normal, the white blood-count was 10,400, red blood-count 4,600,000. On the ninth day a duodenal drainage by Lyons' method was made, and definite *a* bile, *b* bile, *c* bile were obtained. This strengthened our idea that there was no obstruction of the bile passage. Cultures of the bile were negative. On July 15th we noticed marked diminution in liver dulness. The patient was listless and drowsy, took no interest in things. The CO₂ content of blood-plasma was 50.4 per cent volume.

On the 18th, drowsy, could not be aroused, comatose. The patient's coma increased, a hypostatic pneumonia developed, death occurred on the twenty-fourth day after admission. A postmortem examination was held by the corner's physician, and I am able to show you a portion of the liver which Dr McCarthy secured for us. The liver is shrunken in size, it is flabby, its capsule is wrinkled. The cut surface shows the typical raised yellow areas with mottled red areas depressed below the yellow areas. The etiology of acute yellow atrophy is not definite. It is most common in women following pregnancy. It is said to follow acute streptococcus infections, influenza and typhus fever. The relation to acute catarrhal jaundice is an interesting one. Some authors, particularly of the French school, hold that catarrhal jaundice is a benign infective icterus due to changes in the liver cells and not obstructive in character. Recently there has been numerous observations to the effect that such so-called benign infective icterus may be the early stage of acute yellow atrophy, and that severe cases represent the true yellow atrophy. The relation to lues and salvarsan is also an interesting one. Yellow atrophy may occur in secondary lues, and is known to have increased since the introduction of salvarsan. We are entirely at a loss to account for the cause in this case. There is a story of the patient having received some hypodermic injections but the postmortem revealed no changes in any of the other organs which might point to poisoning. It is interesting to note that the clinical opinion of acute yellow atrophy was expressed because of having seen a number of similar cases of severe so-called catarrhal jaundice which progressed to acute yellow atrophy.

CLINIC OF DR HARRY A SINGER

Cook County Hospital

SOME COMPLICATED AND UNCOMMON GASTRIC LESIONS

ALTHOUGH pathologic states of the stomach other than peptic ulcer and gastric carcinoma are met with not infrequently yet only rarely is the preoperative or antemortem diagnosis of the less common morbid gastric conditions confidently predicted. Indeed, in a retrospective survey of the cases followed to the operating and autopsy rooms one is struck by the constancy with which simple ulcer or carcinoma is diagnosed in spite of insufficient evidence or the presence of incompatibilities.

In order to further our acquaintanceship and thereby enhance the more frequent recognition clinically of different types of gastric lesions, I have selected for this clinic 3 cases representing conditions which furnish, as a rule, diagnostic difficulties.

I MULTIPLICITY OF GASTRIC LESIONS—THE IMPORTANCE OF OCCULT BLOOD IN THE STOOL

Clinical History—The first case concerns an individual fifty-three years of age, who, after being observed and treated on the medical service for gastric ulcer during a period of six weeks, was transferred to surgery. The preoperative anamnesis and findings are well summarized in a résumé written into the record of the case by my colleague, Dr Walter L Palmer under whose charge the patient was placed. It reads, "This patient gave a fair ulcer story, one of cramp-like pain in the upper abdomen occurring periodically for the past five to seven years. The exact duration of each attack could not be definitely ascertained, but the pain was present every day for a few days or weeks."

followed by a pain-free period of considerably longer duration. The patient did not strictly localize the pain, but indicated that it was felt across the entire upper abdomen. The distress manifested itself, beginning usually from one to three hours after meals. The effect of food taking and alkalis had not been definitely determined previously, but in the hospital it was found that the ingestion of food (egg-nog), the administration of alkalis, vomiting, and aspiration of the stomach gave complete relief. The introduction of acid into the stomach brought on regularly his distress, which disappeared when the acid was neutralized. In short, it presented the characteristics of a chemical type of pain. During the past six weeks the patient has been on strict Sippy management for peptic ulcer, during which time he has been entirely *pain free* and has *gained 28 pounds* in weight. During the course of treatment the severity of the pain produced by the introduction of acid into the stomach steadily decreased as the length of the period of management increased, so that in the recent final test the patient experienced practically no discomfort whatever.

"The patient is not being referred to surgery because of pyloric obstruction for this has practically disappeared under management, and therefore was probably inflammatory or spastic in nature. The rather frequent vomiting which was complained of upon admission occurred always at the height of the pain, and hence could be attributed to the distress. The deciding factor in recommending surgical intervention is the persistence during management of *chemical blood in the inside of formed stools* upon repeated, careful examinations, in spite of the favorable results (relief from pain, alleviation of obstruction, gain in weight) of therapy. It is on the basis of the finding of blood in the stools under the conditions mentioned that there is diagnosed, in addition to a peptic ulcer, a second lesion, whether associated with or independent of the first, is at this time problematic. The roentgenologic findings of a low-grade pyloric obstruction with indefinite evidence of a prepyloric lesion, the nature of which even after repeated examination is conjectural, forces the diagnosis to rest upon clinical data. On the basis of the

above findings the presence of a peptic ulcer can be confidently predicted associated with most likely a carcinomatous degeneration of a portion thereof or else an independent early malignant neoplasm "

The operation was performed by Dr Karl A Meyer, who resected the distal two-thirds of the stomach, invaginating the duodenum, and uniting the remainder of the stomach with the jejunum through an end-to-side anastomosis. No evidence of involvement by metastases of the glands or other abdominal structures was found. The patient's postoperative course has been uneventful and he has had no complaints whatever.

Surgical Specimen —The resected portion of stomach which has been cut along its greater curvature is, as you see (Fig 280), an unusual one, for in this one specimen are represented three distinct types of gastric disease.

Corresponding to the site on the outside of the stomach of a palpable and visible fibrous thickening, a shallow, oval defect (*a*) of the gastric lining is found situated along the lesser curvature. The margins of this ulcer are quite regular, the edges only slightly raised, and the walls gently sloping. The base is smooth and the floor covered by a thin layer of débris. Converging toward the ulcer are radiating lines, indicating that the process of healing had led to cicatricial changes at the site of the lesion. There are no alterations in any portion of this ulcer, macro- or microscopic, to indicate the presence of a malignant process.

Distal to the point of ulceration and limited exclusively to the pyloric portion, the mucous membrane is thicker and tougher than normal, and presents closely set, circumscribed, verrucous and relatively smooth prominences of various size and height. The warty excrescences are grouped mainly in the immediate prepyloric zone where they form sessile polyps. One such polypoid growth (*b*) with a broad base lies adjacent to the distal end of the oval ulcer. The anatomic picture is that of a verrucous and polypoid catarrhal gastritis limited to the pyloric region, the proximal portion of the resected stomach being free from the above described changes.

If one observes the prepyloric zone closely a very shallow

defect (*c*) hardly more than a mucosal erosion, its depth exaggerated by virtue of the thickened mucous membrane about it, is disclosed. This ulceration which has been inadvertently

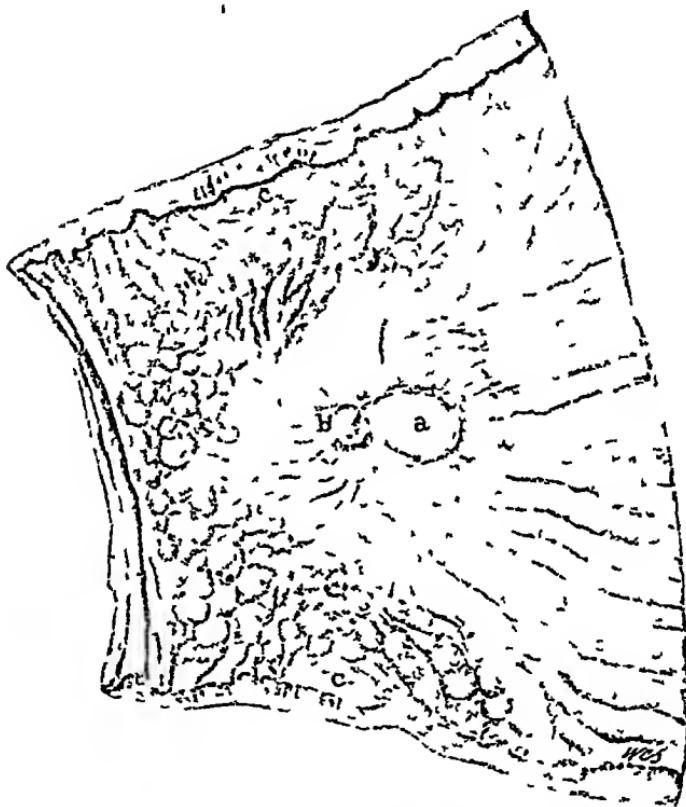


Fig. 280.—Multiplicity of lesions in a resected stomach and first centimeter of duodenum opened along the greater curvature. The irregular mucosal thickenings in the immediate prepyloric zone and along the divided edges represent a polypoid gastritis. One isolated sessile polyp (*b*) is located on the lesser curvature, and distal to it is a healing peptic ulcer (*a*). A second but less prominent indefinitely demarcated defect (*c*) through which the incision has passed is an early carcinoma, superficially ulcerated (see Figs. 281 and 282).

divided in opening the resected portion, measuring in all approximately 1 cm in diameter, has irregular margins and an uneven floor. The consistency and color of the tissue comprising its

edges and base do not differ in any remarkable manner from the surrounding tissues, nor are the gastric layers subjacent to the ulcer definitely fixed to each other. On the cut surfaces the base instead of being, as in a peptic ulcer, smooth, homogeneous and glistening is here rather rough granular and opaque. A microscopic preparation made to include the entire ulcer shows normal mucous membrane at each edge with large



Fig 281.—Margin of the ulcerating, superficial lesion (*c*) pictured in Fig 280. The glandular elements are atypical in appearance (see Fig 283), but do not penetrate the muscularis mucosæ. A marked inflammatory cell reaction is present.

atypical glands at one margin (Fig 281) adjacent to which the tissue takes on the appearance of a scirrhus (Fig 282). Here the acini scattered in a fibrous stroma rich in collagen fibers are smaller and exhibit a marked deviation from the normal both as to form and relationship. The individual cells comprising these glands likewise present atypical characteristics. The glandular elements in the section examined are seen to

break through the muscularis mucosæ to invade the submucosa, extending to, but not involving the muscularis. The surface of the growth is superficially ulcerated and the subjacent tissues infiltrated by inflammatory cells. The unanimous opinion of several pathologists who have examined this section is that it represents an early carcinoma. In histologic preparations from various other portions of the resected stomach, including several

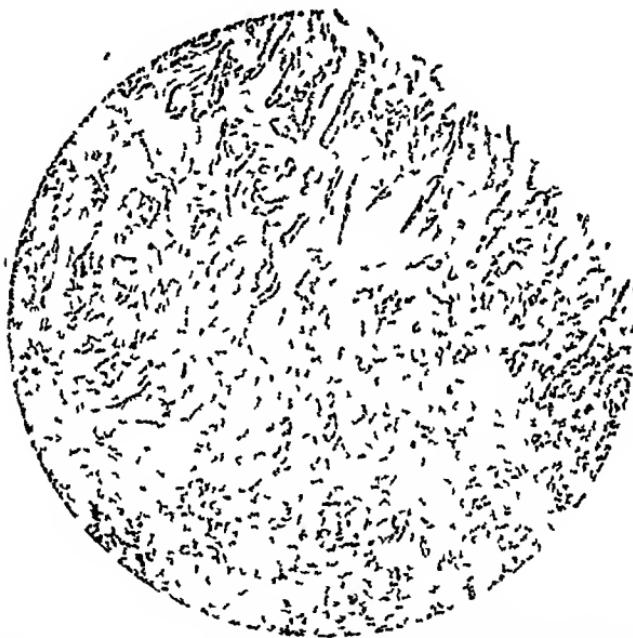


Fig 282.—Carcinomatous proliferation of the gastric glands with invasion of the submucosa. From the central portion of the same microscopic section of which Fig 281 forms the peripheral part.

of the polyps, no evidence of carcinomatous growth can be found. So-called precancerous changes—*i.e.*, the presence of rather atypical proliferated glandular elements with cells containing hyperchromatic nuclei—are frequently encountered (Fig 283), but the significance of these structures is as yet not established.

Comment—The anatomic changes in the stomach explain quite adequately the complexity of clinical findings, for it can be assumed that the therapeutic management led to reparative

processes in the peptic ulcer, and consequent disappearance of the pain and vomiting, but failed to influence the superficial carcinomatous ulcer which was responsible for the persistent blood in the stools. That the importance attributed to this solitary finding of chemical blood in the stools in repeated examinations, in spite of the apparent response to therapy, was quite justifiable is borne out by the operative findings. The

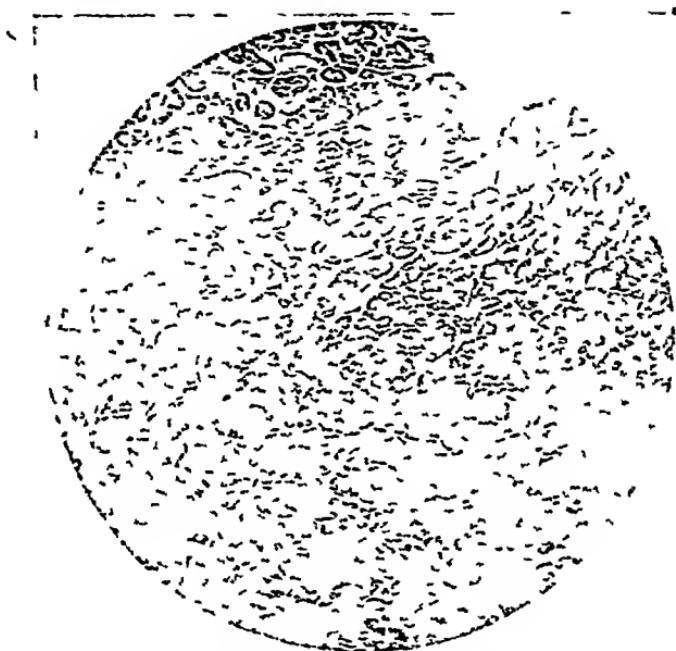


Fig 283.—Adenomatous hyperplasia in a polypoid excrescence of the mucosa. The somewhat atypical appearance and distinct hyperchromatism involving the acini in the upper left corner of the section suggests a resemblance to the glands in Fig 281.

value of the test for occult blood (assuming, of course the usual sources of error are eliminated) in gastro-intestinal disease as illustrated in the foregoing case indicates its more frequent utilization.

Although the polypoid condition played no direct role in the clinical picture nevertheless a consideration of its possibilities might be of value in that polyps unless portions thereof are

aspirated, almost always elude recognition *in vivo*. It should be remembered that the tendency toward ulceration of benign polypoid growths which has been known for a long time not infrequently leads to the presence of blood in the feces. Whether the possibility of a polyposis was considered to account for the persistent chemical blood is not stated, but the relative frequency of carcinoma and the age of the patient (fifty-three years) would be weighty factors in favor of the diagnosis of malignancy. Although gastric polyps when situated in the region of the pylorus may give rise to cramp-like pain and the symptom-complex of pyloric stenosis (Ledderhose¹), there is no basis for incriminating them in this case, for the pain as demonstrated so clearly by Dr. Pahner was of a chemical nature, whereas the obstructive phenomena were practically entirely relieved by ulcer management. A retrospective study of the x-ray films discloses in the prepyloric zone an occasional area slightly smaller than a cherry seed, somewhat circumscribed, and a trifle less radiopaque than the surrounding gastric shadow, requiring, however, extremely close examination for its discernment.

From the standpoint of genesis and the interrelationship of the various morbid processes found in this stomach many interesting problems can be evolved, a discussion of which, however, would carry us too far. Whether or not the presence of the peptic ulcer antedated and produced the catarrhal gastritis, or whether the latter was pre-existent and a factor in the development of the former, is highly conjectural. The association of the carcinomatous ulcer and the hyperplastic polypoid mucosa of the stomach is, on the other hand, a definite sequential one, for the tendency toward malignant degeneration is characteristic of mucous polyps, occurring in 50 to 60 per cent of the cases (Albu²). According to the same writer, polyposis forms the second most important precarcinomatous morbid condition of the stomach peptic ulcer being the first. (Figures 283, 281, and 282 illustrate in the order named progressive degrees of variation of glandular elements from the normal, and may represent graphically successive stages in the transformation of an adenomatous polyp into an adenocarcinoma.) It seems, therefore,

desirable that if in a stomach in which at operation in addition to another gastric lesion a polyposis limited to the pylorus (which is the more frequent localization) is found, a gastric resection should be performed. Since in the case presented the polypoid state was well within the limits of the resection a most favorable outcome made possible through careful stool analysis and clinical interpretation together with judicious surgery, is anticipated.

II LINITIS PLASTICA—LEATHER-BOTTLE STOMACH

Clinical History—The second case is that of A. P., a white male also fifty-three years of age, who complained upon entrance of epigastric pain, vomiting, constipation and the loss of weight all of one month's duration. The pain was of a sharp character more or less continuous, and aggravated by eating. Emesis at the onset of the disease, did not occur until at least several minutes after the intake of food. The vomitus at that time consisted apparently of gastric contents, since ingested milk, for instance was returned in curds. As the course of the disease progressed however the vomiting would follow in an increasingly shorter period of time so that during the few days prior to admission ingested food was returned within a minute or two. The loss of weight during the month's illness was estimated at 20 pounds, constipation of an extreme degree was present during that time.

The essential physical findings consisted of a marked degree of emaciation and obvious weakness, a scaphoid abdomen with a slight fulness in the epigastric region, where a sense of resistance and a questionable smooth firm rounded mass was encountered. The patient was requested to swallow as much water as possible and voluntarily limited the amount to one mouthful. After about thirty seconds singultus occurred, immediately followed by regurgitation of the fluid. Using milk in a second test, the patient swallowed 4 ounces which was regurgitated in part within fifteen seconds, the remainder following within the next minute. None of the returned milk was coagulated.

On the basis of the anamnesis and the above observations

Dr Lawrence Jacques assumed an extensive lesion of the stomach leading to extreme diminution of its capacity to be present. Dr Jacques reasoned that since the patient a short time previously had vomited coagulated milk, it was evident that the food ingested then came in contact with the gastric juice, and he excluded therefore an obstruction at or above the cardia. The failure of the milk to curdle at the present time he interpreted to be due to a further stage of the original process, leading to the inability of the stomach to retain even small amounts. The diagnosis submitted before the patient was examined fluoroscopically was leather-bottle stomach probably as a result of diffuse carcinomatous involvement.

Since the degree of emaciation and dehydration demanded immediate action, the esophagus and stomach were examined roentgenologically and the patient prepared for operation shortly after the entrance examination was made. The x-ray report submitted by Dr C. A. Matthews reads, "There was noted an extreme delay in the passage of the barium mixture to the distal esophagus. The lower end of the esophagus was distended to a width of $2\frac{1}{2}$ inches supporting a column of a smaller diameter reaching to the pharynx. The proximal stomach distended to walnut size, beyond which was found a narrow filament about 3 inches in length, followed by a large thimble-sized barium shadow which may be pyloric extremity or small duodenal bulb. If the stomach has not been subjected to an operative procedure, a contracting lesion such as scirrhouous carcinoma may be presumed to cause almost complete obliteration of the cavity of the stomach."

A laparotomy performed under local, supplemented by nitrous oxide, anesthesia disclosed an extremely shrunken stomach which formed a sausage-shaped mass in the upper abdomen. On account of the almost complete obliteration of the gastric lumen a jejunostomy was performed. The postoperative course was uneventful until the jejunostomy tube broke loose from its attachments and required reinsertion. Following replacement of the catheter the patient went rapidly down hill and died with signs of a generalized peritonitis.

Morbid Anatomy—The enterostomy opening was collapsed, and the tube which had recently been replaced was found lying free in the abdominal cavity with a resulting diffuse fibrinous purulent peritonitis, the immediate cause of death. There were



Fig. 284.—*Linitis plastica*. The extreme shrinkage involves the entire stomach. The proximal half of the stomach is laid open to expose the lumen and the cut section of its wall. The thickened submucosa from which fibrous strands pass into the hypertrophied musculature is best seen just beyond the cardioesophageal junction (c). A portion of duodenum (d) is attached.

other changes in the body associated with and independent of the acute infection but of no direct relationship to the gastric pathology.

The stomach is indeed an unusual one from the standpoint

of size (Fig. 284) Its description and also that of the esophagus dictated at the necropsy reads, "This stomach measures from the pylorus to the cardia along the greater curvature 16 cm, along the lesser curvature 11 5 cm The organ is smooth outside and extremely shrunken, forming a sausage-shaped, semi-circular mass, with greatly thickened walls When opened along the lesser curvature its lumen is seen to be greatly diminished its mucosa smooth and intact, and its walls thickened, due to an increase in the width of the submucosa and also of the muscularis The thickness of the wall at the esophageal end is 7 mm, at the pyloric end, 8 mm There is an abrupt thinning of the wall of the pyloric end of the stomach as it passes over into the duodenum The transition at the cardio-esophageal junction is likewise distinct, the process being limited quite definitely to the stomach Although the cardio-esophageal and pyloric orifices are patent, there is significant narrowing which resists dilatation There are three enlarged lymph-glands along the greater curvature of the stomach which are pink, fleshy, and grossly free from tumor tissue "

The microscopic study of sections from many parts of the stomach and from the perigastric lymph-glands after careful search fails to show any distinct evidence of carcinoma The outstanding changes in the stomach (Fig. 285) are an atrophy of the mucosa, a striking dense, fibrous thickening of the submucosa, and extension of connective-tissue bands through the muscularis The lymph-glands are altered by focal fibrous replacement of the normal structures

Comment—In regard to the symptomatology of leather-bottle stomach a great deal can be written Those manifestations which have as their basis a progressive decrease in the size of the gastric lumen are the only ones however, which are characteristic of the disease Of the greatest importance in the history is the symptom of vomiting the nature of which can be readily deduced from a knowledge of the underlying pathology In the early stages of the disease emesis is of little consequence, occurring rather infrequently As the capacity of the stomach diminishes, however the vomiting or regurgitation becomes

more and more frequent being excited by the ingestion of progressively smaller amounts of food, until finally intolerance to an excess above a surprisingly small amount of fluid develops.

The course and character of the vomiting described above may be closely simulated by an esophageal constriction, a fact of which the examiner of today's case was fully cognizant. In order to locate the site of obstruction Dr. Jacques made special



Fig. 285.—*Linitis plastica*. The mucosa is atrophied and the submucosa greatly thickened by virtue of a fibrous increase. The smooth muscle-fibers are cut transversely. A lymphatic vessel (*a*) filled with round cells might be looked upon with this magnification as carcinomatous.

inquiry into the nature of the vomitus during the earlier stages of the disease a consideration of much diagnostic importance. The patient affirmed repeatedly that ingested milk formerly was returned in a coagulated state, indicating that the milk reached the stomach and that the obstruction was, therefore, beyond the cardia a conclusion borne out by subsequent findings.

The presence of a sausage-shaped tumor lying transversely in the epigastrum is not an uncommon physical finding in the later stages, but more frequently only a sense of resistance is encountered. The degree of motility of the shrunken organ is dependent upon the presence of adhesions, which in this instance were absent. The position of various diagnostic methods dealing with the injection of water or inflation of the stomach has been usurped by the more enlightening roentgenologic examination. The evidence adduced through this means is by far the most valuable in the diagnosis of the condition for the diminutive lumen, the rigid walls (indicated by absence of peristalsis and incompressibility of the organ), the patent pylorus, and the rapid emptying, in spite of which the esophagus remains filled, are usually demonstrable.

Under the term "linitis plastica," or leather-bottle stomach, a variety of processes leading to a contracted state of the viscera have been included (see Lyle³). Where unmistakable malignant epithelial elements are conspicuous the genesis of the condition is clear, and the presence of a scirrhouous carcinoma easily established. There is a group of cases, however, in which the histologic distinction between inflammatory and neoplastic contracture of the stomach is extremely difficult to draw. The fact that the carcinoma cells may be reduced to widely scattered islands or even to isolated cells in the dense connective-tissue stroma has probably led to the inclusion of many scirrhouous carcinomas in the benign group. The absence of malignant cells over considerable areas of the stomach in these cases has led to the assumption that linitis plastica is, in the majority of instances, the result of a healed or almost healed disseminated scirrhouous carcinoma. Ewing⁴ concludes, "although the existence of miscellaneous forms of purely inflammatory gastric and pyloric stenosis cannot be denied, it now appears, largely through the critical analyses of Bret and Paviot, Meinel, Krompecher, and Makai, and others, that the great majority if not all the cases of linitis plastica are atypical fibrocarcinoma."

In our case a careful search for traces of carcinoma was instituted, serial sections of blocks of tissue from the stomach and

perigastric lymph-glands being examined with great pains An occasional group of round cells, collected in a lymphatic vessel (Fig 285, a) gives the general appearance of a small spheroidal-cell scirrhus, but under higher magnification the cells present the morphologic characteristics of lymphocytes In many other places closely grouped cells suggesting the presence of heterotopic epithelial nests are, upon closer examination, seen to be proliferated endothelial and connective-tissue cells Although the inclination from the microscopic side is to look upon today's case as benign, yet the possibility of undiscovered or obscured malignant elements is to be considered as well as the personal factor in the interpretation of the proliferated cells found

In his excellent article on the subject, Lyle³ differentiates between the benign *limitis plastica* (diffuse fibrosis) and the malignant, stating that the diagnosis of the benign form is rarely possible and always problematic It should be borne in mind that unless a specific etiologic factor (syphilis, diffuse phlegmonous gastritis, chronic venous stasis) can be demonstrated clinically to account for a benign contracture, or distinctive and associated findings (metastases, etc) to indicate the presence of a carcinoma, the preoperative or antemortem diagnosis as to the type of leather-bottle stomach is highly conjectural

III LYMPHOSARCOMA INVOLVING THE STOMACH

Clinical Record—E N, a white male, entered this hospital for the first time four years ago, at which time he was fifty-four years of age He sought admission on account of swellings of the neck, the largest of which had its origin two years previous This tumefaction, after having been present for two months, disappeared spontaneously, to reappear after an interval of eight months At the time of its recurrence fourteen months previous to entrance the tumor was of hazelnut size, the growth having been progressive since that time Simultaneous with the reappearance of the original tumor similar but smaller swellings were noted, these also having steadily increased in size At no time had he experienced any discomfort other than a feeling of heaviness In an inventory of symptoms by systems the only

additional noteworthy information was the loss of weight, approximated at 25 pounds, during the year preceding entrance to the hospital. The past history disclosed the fact that shortly after the primary recurrence the patient had undergone a throat operation, the nature of or indication for which could not be ascertained. The history of a treated syphilis was obtained, there was no known tuberculosis in the family.

In the physical examination there was disclosed in the anterior and posterior triangles upon the right side of the neck several enlarged lymph-glands, freely movable, discrete, elastic, and painless to pressure, the largest of which being equal in size to a walnut. A slight adenopathy at the angle of the jaw on the left side was also present. No lymphatic enlargements were noted elsewhere except for palpable superficial inguinal glands which were considered of insufficient size to be of significance. The mediastinal dulness was not increased in extent, no abdominal masses were palpated, nor was the liver or spleen felt.

The laboratory tests at the time furnished the following information. Blood Wassermann anticomplementary in two separate tests, spinal fluid Wassermann negative, red cell count 4,960,000, white cell count 12,200, of which 56 per cent were neutrophilic polymorphonuclear leukocytes, 33 per cent small lymphocytes, 9 per cent large lymphocytes (including probably monocytes), and 2 per cent eosinophils. Roentgen-ray examination of the chest exhibited no increase in the mediastinal shadow, the stomach, duodenum, and colon were roentgenologically negative. No biopsy was performed.

The patient was discharged from the ward service after two weeks with a diagnosis of Hodgkin's disease and referred to the out-patient department for α -ray therapy.

The patient returned recently, approximately four years after his first entrance, with the information that following α -ray therapy the cervical swellings had disappeared and that he had remained well up until eighteen months ago, when he perceived a gradual but progressive loss of weight amounting to 55 pounds. Concomitant with the decrease in weight there occurred a persistent weakness, so that during the past two months he has been

unable to work. For the past year he has suffered from pain in the upper abdomen, at its beginning intermittent in character, but recently more or less constant at no time, however, related to food taking or bowel movement. A mass was noticed by the patient in palpating his abdomen about one year ago. This at first was felt to be egg sized but has doubled in size since it was first perceived. At no time has he vomited.

The patient upon examination presented the usual signs of cachexia. In addition, an abdominal mass located in the left upper quadrant of indefinite outlines, firm not tender nor possessed of respiratory mobility was found. In both groins were tumefactions one of these on the right side being as large as a hen's egg. The swellings were nodular elastic not attached to the skin, but lacked independent mobility. No cervical adenopathy was present at this time.

As regards the laboratory side, the blood-picture was that of a high-grade secondary anemia with a normal white count, the test-meal revealed no free or organic acid, the motor meal showed no retention. Stools were ordered to the laboratory but none were obtained before death. An x-ray examination was scheduled and a gland removed for histologic and bacteriologic study, but before these examinations were completed the patient came to autopsy with the tentative clinical diagnosis of an abdominal malignancy or, as the next most likely underlying condition, Hodgkin's disease.

Autopsy Findings.—The postmortem performed shortly after death disclosed upon the external examination, in addition to the generalized anemia and the emaciation, a distinct enlargement of the inguinal glands, bilaterally, but no adenopathy of the superficial lymph-glands elsewhere. The abdominal tumor palpated during life consisted of a gastric neoplasm located on the greater curvature firmly agglutinated to a mass of coalesced perigastric lymph-glands, the latter intimately bound to the tail of the pancreas which, in turn was invaded by tumor tissue. The perigastric structures were involved apparently by direct extension while neoplastic substance passing almost imperceptibly over into the normal structures. The left adrenal gland

was invaded by the same type of tissue, the extension to it being easily traced in a continuous line from the stomach and periaortic glands. The mediastinal glands and the contiguous portion of the left lung were similarly involved by the same type of tumor tissue seen elsewhere. A direct line of glands could be traced along the entire extent of the abdominal aorta, continued along the internal iliac and the first portion of the femoral vessels. Everywhere the tumor tissue was white, uniform in appearance, opaque and resilient.

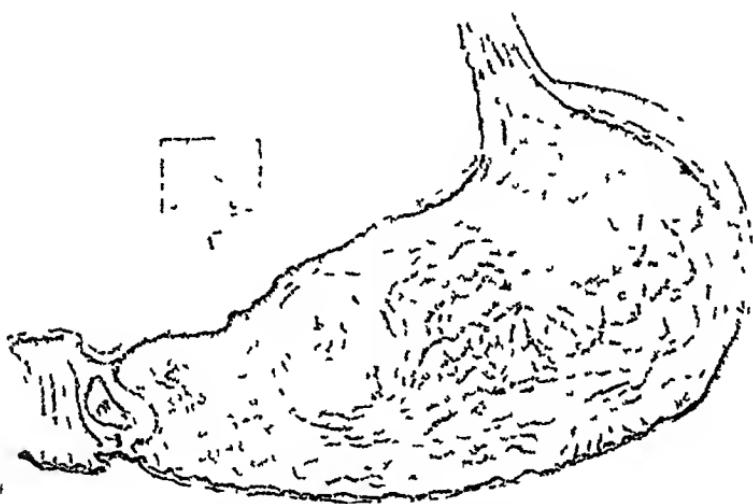


Fig. 286.—Lymphosarcomatous involvement of the stomach. The tumor masses (*a*, *b*, and *c*) have the form of infiltrates. The serous covering over the region of *c* is studded with millet-seed-sized nodules (*A*). Infiltrate (*a*) is deeply excavated and almost completely encircles the pyloric ring.

The stomach, as you see (Fig. 286), is moderately increased in size and irregularly involved by tumor infiltrates. Corresponding to the site of the largest of the neoplastic masses there are studded on the serosa large numbers of closely set opaque millet-seed-sized bodies (*A*). The chamber of the opened stomach is seen to be more spacious than normal, the infiltrates failing to encroach upon the lumen of the stomach, but rather lending a firmness to its stretched walls. In passing from the

esophagus distally the first tumor mass encountered (*c*), located on the greater curvature extending mainly on the posterior wall, and projecting in an exogastric direction is a broad circular, undulating growth 12 cm in diameter, thickest in its central portion, becoming thinner passing centrifugally. The tumor tissue is resilient, and on section opaque, moist, a pure white and of homogeneous structure. The second infiltrate (*b*), which is 8 cm in diameter, is found distal to the first, and, with the exception of its flat surface answers to the same description. The pyloric ring is invaded in all but 1.5 cm of its circumference by a third infiltrate (*a*), which differs from the previous two in that it is deeply excavated, forming an ulcer with margins and base consisting of tumor tissue similar to that described above. In spite of the presence of this lesion, before the pyloric region was incised, a noteworthy degree of stenosis was not demonstrable.

The microscopic preparations of tissue from various places present the characteristic features of a lymphosarcoma, the infiltrative properties of which are well illustrated in this section of pancreas (Fig. 287).

Comment—This case is particularly unique for purposes of instruction in that it embodies in its clinical and anatomic manifestations an unusual number of the classical characteristics of lymphosarcoma and inasmuch as a knowledge of its natural history, general as well as local is most essential for the recognition, clinically, of the gastric phase of the disease a recapitulation of the representative features of lymphosarcoma as related to the case at hand, may prove of value.

Lymphosarcoma in General—Basing his conclusions upon a study of 50 cases of lymphosarcoma and double the number of related cases Kundrat⁵ separated this disease from the other aleukemic lymphatic tumors through not only anatomic characteristics but also clinical features. He pointed out that these tumors are connected genetically with the lymphatic apparatus, arising however, not only from lymph-glands but also from adenoid tissues elsewhere as in mucous membranes. The growth does not remain limited to the site of origin but sooner or later extends beyond the capsules of the glands and boundaries of the

follicles, leading to a coalescence of the individual elements and infiltration of the surrounding tissues. In this regard the lymphosarcoma differs from other primary lymph-gland tumors, as leukemia and pseudoleukemia, in which, although the glands may become coalescent, they do so by virtue of compression of their capsules. In the case being presented today the primary growth had its origin presumably in the cervical glands, but under

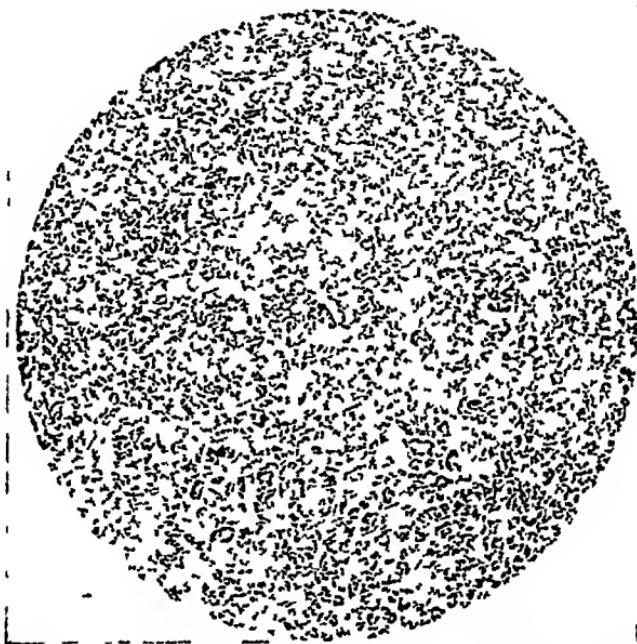


Fig. 287.—Lymphosarcomatous invasion of the pancreas. The aggressive nature of the growth is evidenced by the wide separation of the pancreatic acini and islets by infiltrated tumor cells.

the influence of the Roentgen ray the neoplasm had entirely regressed. As pertains to the stomach, whether the involvement here represents a secondary spread or the site of the primary growth cannot be unequivocally established. In either event the origin of the tumor is obviously in the lymphatic apparatus. The intimate relationship of the individual nodes along the greater curvature of the stomach and the obliteration of their outlines bear witness to the marked tendency of this growth

to spread beyond the fibrous capsules of the glands. The extension from the stomach to the perigastric glands (or perhaps the reverse) and the involvement of the pancreas and left adrenal illustrate further the infiltrative property of the lymphosarcoma.

Kundrat maintained, furthermore, that lymphosarcoma is essentially a regional disease remaining restricted to several regions, spreading by direct propagation through the lymphatics to the neighboring glands or follicles and never becoming generalized as in leukemia and pseudoleukemia. In solid tissues the extension is along the lines of least resistance in a tubular structure with a mucous membrane lining for instance along the submucosa the infiltration spreading in a circular manner, as in carcinoma but expanded over a greater area, leading to widening rather than stenosis of the lumen. Our case conforms to this regional conception in that only certain chains of glands are implicated whereas the axillary, mesenteric, anterior mediastinal, and other groups which are frequently involved in a generalized process are spared. The gastro-intestinal involvement is likewise strictly regional. Although only the pyloric growth encompasses the lumen of the stomach, the other two infiltrates are diffuse in character and tend to spread in a circular fashion and in spite of this, no narrowing is observed.

In addition to the original tumor and its local and regional extensions there occurs, according to Kundrat in distant organs similar growths assuming the characteristic form of diffuse multiple infiltrations which differ from ordinary metastases, as those occurring in carcinoma and true sarcoma in not being nodular. Furthermore, a careful search will indicate the presence of a continuous lymphatic connection between the distant growth and the primary site in that the lymph-glands between the two are similarly affected. Actual metastases which can be explained only by propagation through the blood-stream are isolated and rare, due to the fact that the growth tends to compress the blood-vessels. The presence of many of the remote formations can be explained by retrograde lymphatic extension rather than by hematogenous route. The gastric tumor masses

in today's cases exemplifies, according to the above description, the characteristics of secondary growths being remote from the original source, and having the form and appearance of infiltrates rather than nodules. The extension by permeation of the lymphatics can be traced from the primary site, presumably the cervical region, in a continuous chain through the posterior mediastinal to the upper abdominal, and in a further retrograde manner to the lower abdominal, iliac, femoral, and inguinal lymph-glands.

The secondary affections, the same writer asserts, occur almost exclusively in the gastro-intestinal mucous membranes and serous coats, the former being a site very rarely involved in the most wide-spread and luxuriant types of neoplasms as true sarcoma and carcinoma. In contrast to leukemia and pseudoleukemia one never sees involvement of the spleen and liver, which unless altered by extraneous processes, are actually decreased in size (due to marasmus). A slight diminution in size of the liver and spleen was noted also in our case, another point in common with the general features of lymphosarcoma. Since there are so many important points of similarity between Kundrat's description of the disease and today's case, the latter appears particularly well suited to serve as a model to illustrate the specific characteristics of lymphosarcomatosis.

Lymphosarcoma of the Stomach.—In regard to lymphosarcoma as it affects the stomach itself there are a number of distinctive local features in addition to those mentioned in the general discussion of the disease that are noteworthy. Kundrat calls attention to the observation that infiltrates are not predominantly located in the pyloric portion, a finding corroborated by others. Sternberg⁶ comments on the almost universal rule of dilatation of the stomach in the presence of lymphosarcoma, while Ruffin⁷ emphasizes in his category of clinical features of diagnostic value the absence of pyloric stenosis even though the ring may be encroached upon. Peculiarly enough the stomach being demonstrated presents these three important characteristics of gastric lymphosarcoma, for the greatest involvement lies toward the fundus, the capacity of the stomach is increased, and the

pyloric lesion leads to no significant degree of stenosis (clinically the patient did not vomit nor did the motor meal show retention) These changes might have led to a suspicion of the true nature of the lesion had the patient been fluoroscoped The stiffness of the walls over a considerable portion of the gastric silhouette preventing peristaltic contractions would be further evidence favoring the diagnosis of lymphosarcoma

The diagnosis however, is at best difficult unless the condition is not restricted to the stomach and regional glands A noteworthy feature in this case which might have aided in the diagnosis even before a biopsy was resorted to was the finding of enlarged inguinal glands Based upon the associated presence of a well-marked superficial inguinal adenopathy the prediction can be (and actually was) made that a known gastric tumor is likely to be of lymphatic nature (lymphosarcoma, pseudoleukemia or Hodgkin's disease), since carcinoma rarely metastasizes to these nodes

It is generally stated that a fair percentage of the reported cases have occurred in individuals under the carcinoma age In our instance this factor would not have assisted in the diagnosis, since the patient was fifty-five years old Of the other clinical features which may aid in a diagnosis are the absence of cachexia until a very late period, and the response in some cases to arsenic, α -ray therapy, acute infections, or spontaneous disappearance of the swellings The absence of anemia and emaciation until late were noteworthy in our case, as was also the immediate retrogression of the cervical swellings following exposure to the Roentgen rays

The question of primary or secondary lymphosarcoma of the stomach is often debatable, for the frequent associated involvement of neighboring lymph-glands renders it difficult to ascertain whether the gastric mucous membrane or the upper abdominal lymph-nodes gave rise to the original growth A great number of the cases in the literature reported as primary in the stomach are probably secondary, since the gastric tumors are part of a diffuse regional process It is generally stated that the primary growths occur mainly in the form of wide-spread in-

filtrates even involving almost the entire stomach, whereas the secondary growths are more frequently multiple. The number of lesions in the stomach presented (three) supports the assumption based upon the clinical history that the gastric involvement was secondary, since the cervical enlargement attracted attention four years before gastric symptoms became manifest. Furthermore, roentgenologic examination of the stomach during the patient's first stay in the hospital in 1921 failed to disclose any lesion.

Prognosis of the Disease.—Kundrat considered lymphosarcoma even more malignant than carcinoma or true sarcoma, and, indeed, designated it as a true *non-me-tangere*. He recognized that scarring and shrinkage might follow ulceration, but that healing was only partial, since simultaneously the disease often progressed elsewhere in the individual's body. Extirpation of the involved structures even if performed early, according to the same writer, was often of no avail, but, on the contrary, might act as a stimulus to spread. Since Kundrat's work there have appeared in the literature a number of reports in which spontaneously or following surgical intervention the application of radient rays, the use of arsenic internally, or after acute infectious disease, the process disappeared. However, in most instances the tumor recurred elsewhere and led to a fatal outcome, as is true in our patient. Eisenmenger⁶ describes a case in which a biopsy was the cause of the ulceration that led to almost complete disappearance of the tumor and an alleviation of symptoms. Lewing⁷ found in a fatal case with gastro-intestinal metastases the original ulcerative lesion of the sinus laryngis completely healed. In our own case the throat operation performed in 1920, five years previous to the patient's death, may have been part of the same disease process, but, unfortunately, no specific information regarding the procedure or the indication therefore is available.

According to Russin,⁷ the only case of cure on record is that of Ruppert.⁸ The latter gives an interesting report of a woman seventy-two years of age in good health, in whom he had performed a subtotal gastrectomy for lymphosarcoma fourteen

years previously. However, as he himself states, this is extremely exceptional, for in no other case on record did the patient survive the operation for as long a period. Our own case is instructive from a prognostic standpoint also, since it illustrates the fallacy of considering a case of lymphosarcoma as cured upon the basis of the elimination of the only demonstrable lesion, for recurrences, which are the rule, should be generally expected even after a relatively long quiescent period.

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CLINIC OF DRs WILLIAM A BRAMS AND
E P WOLF-BLITZSTEN

MICHAEL REESE HOSPITAL

CLINICALLY DORMANT CARCINOMA AND THE IM-
PORTANCE OF A COMPLETE PHYSICAL EXAMINA-
TION PAROXYSMAL TACHYCARDIA

THE first patient we wish to present today illustrates the clinically quiescent manner in which carcinoma may exist without producing symptoms suggesting either its presence or location. It may truthfully be said that carcinoma may come "like a thief in the night" to sap the life and strength of its unaware victim until the condition is discovered in so advanced a stage that help is no longer possible. This case also brings out the point that malignancy should be suspected in patients suffering from anemia which cannot be explained on another basis and which occurs in persons of middle or advanced age.

The patient, P R, a freight handler, forty-eight years of age, was informed by his physician that he was suffering from anemia and came to us for treatment because a course of management for three months failed to provide improvement. He complained of some shortness of breath on exertion and felt a little weak, but had no other symptoms. He had lost a few pounds in weight, but he had no symptoms referable to the gastro-intestinal tract, except occasional belching and heartburn. There was nothing of importance in his past or family history and his habits were temperate.

Examination revealed a middle-aged patient in a good state of nourishment. There was a distinct yellowish tinge to the skin and moderate pallor of the mucous membranes could be seen. No jaundice was found. There was nothing abnormal in the heart or lungs, and careful palpation of the abdomen failed to reveal anything abnormal. Rectal examination showed

nothing pathologic. The blood Wassermann was negative, as was the urine. The blood showed 3,100,000 reds, 60 per cent hemoglobin, 12,400 whites, and a normal differential count. A stained film of the blood showed many nucleated red cells, poikilocytes, and some macrocytes and macroblasts. The stomach contents, after an Ewald test-meal, showed free HCl 10 and combined acidity 32. No long bacilli were found. The stools showed a faint trace of blood with benzidin, but none with the Weber test.

Comment and Outcome—A study of the patient at this time gave us little information except that he had a pronounced anemia. No evidence was obtained pointing to a possible cause for this anemia, and on first thought the large number of nucleated red cells and poikilocytes together with the macroblasts pointed to pernicious anemia. The stained blood-film looked very much like that of pernicious anemia, but there were certain other features which led us to abandon this diagnosis. The color-index of 0.95, while not positive evidence against pernicious anemia, was, nevertheless, somewhat low for this condition. Very strong evidence against pernicious anemia was the slight leukocytosis of 12,400, which is in marked contrast to the normal count or actual leukopenia characteristically found in pernicious anemia. Another possibility to be considered was infection with the fish tapeworm, which is not uncommon in the region of the Great Lakes, and which causes a blood-picture closely resembling pernicious anemia. Numerous stool examinations failed to reveal any evidence of parasites, and it was concluded that the patient was suffering from a severe secondary anemia with a positive effort at regeneration of red cells.

Our attention was now directed to the possibility of malignancy in an early stage and before localizing symptoms would have a chance to show themselves. The history and the results of the physical examination furnished no clew to the possible location of such a malignancy, but it was decided to concentrate our attention on the most frequent localizations of malignancy in the male. An α -ray examination of the stomach was, therefore, made in spite of the fact that the gastric contents showed

no great deviation from the normal, and the fact that the stools contained no blood with the Weber test Much to our surprise, a large filling defect was seen at the pylorus extending for a distance of about 5 cm, so that a thin and irregular channel replaced the normal pyloric part of the stomach There was no tenderness over the filling defect, but examination six hours after ingestion of the barium meal showed a residue of at least three-fourths of the meal A diagnosis of infiltrating carcinoma of the stomach was made and an exploratory operation recommended in view of the absence of all symptoms and the possibility that the neoplasm was still in an early stage so that resection could be performed On opening the abdomen a large carcinoma was found extending along the lesser curvature from the cardia to the pyloric orifice There was marked glandular involvement, but no metastasis in the liver The stomach was adherent to the neighboring structures and could not be resected, so that only a palliative gastro-enterostomy was performed

The second case is of interest because the anemia in a middle-aged woman was discovered accidentally, the patient complaining of congenital heart disease She was not aware of anything except dyspnea on exertion and precordial pain, and made her own diagnosis of a heart lesion, being completely unaware of the more dangerous lesion in her rectum This case is of further value in that it emphasizes the point that no examination is complete unless the rectum is carefully examined regardless of the symptoms and signs found

The patient Mrs H G, fifty years of age, entered the medical service of Dr Solomon Strouse at the Michael Reese Hospital with a complaint of congenital heart trouble She stated that she had this trouble since childhood, and that for the past three years she had precordial pain and dyspnea on exertion She noticed that her abdomen had become swollen during the past five weeks

Examination revealed a pale, middle-aged female who had slight dyspnea but no cyanosis The apex of the heart was diffuse and in the fourth and fifth interspace No thrills were palpable The left heart border was 12.5 cm and the right

35 cm from the midline. A loud systolic murmur was heard at the apex and base and was definitely transmitted to the left and right. The aortic and pulmonic second sounds were not definitely made out. The liver was enlarged and tender and free fluid was found in the abdomen. The Wassermann was negative, as was the urine. The blood-pressure was normal and examination of the blood showed 5,600,000 red cells, 65 per cent hemoglobin, and 10,500 white cells, with a normal differential count. The stools were negative for blood on eight different examinations. A diagnosis of decompensated heart was made, and the pallor suggested that some infection was still present in the endocardium. It was necessary, however, to rule out pernicious anemia and malignancy before accounting for the pallor on the basis of an infectious endocarditis. A vaginal examination made by Dr. Jacob Meyer revealed a mass on the posterior wall which proved to be a cauliflower carcinoma of the rectum. Proctoscopic examination confirmed the diagnosis. The finding of this carcinoma is of particular interest because there were no symptoms attributable to this region, no constipation or diarrhea was present, no blood was found chemically on eight different occasions, and still the lesion was very large and easily found, providing an examination of the rectum were made. It is obvious that of the two conditions from which this patient suffered, the clinically dormant rectal carcinoma was of infinitely greater importance than the clinically apparent heart decompensation which the patient herself diagnosed.

TACHYCARDIA

Tachycardia includes all of those conditions, excepting pyrexia, in which the rate of the heart is regular and abnormally rapid, beating from 100 to 200 beats per minute. When the rate exceeds 200 beats per minute the condition is generally considered auricular flutter.

When the increase in the heart rate is gradual, the transition from slow to rapid and from rapid to slow being so gradual as to be unobservable, and when the increased rate is more or less constant, lasting any length of time from a few minutes to many

months, we call it simple tachycardia. When, on the other hand, the onset and the end of the rapid rate is very abrupt, and the observer and even the patient can detect the sudden transition in rate, we speak of paroxysmal tachycardia.

Simple tachycardia may be due to physiologic acceleration of the heart, and occurs when outside demands on the heart are more than usual. This may take place following exercise or emotion. In certain types of individuals, particularly those with an unstable nervous system, there may be a tachycardia accompanied by sensations of distress following very slight exertion.

There is a conspicuous and sudden increase in heart action following a relatively slight disturbance in certain types of individuals, namely, those with exophthalmic goiter. This is the most frequent and constant cause of tachycardia clinically speaking. In these cases the pulse is regular and the rate may be anywhere from 100 to 200 beats per minute. In this condition the tachycardia is accompanied by definite evidences of arterial change, such as flushing and paling of the skin, local sweating, and often visible pulsation in the superficial arteries and veins. The exact cause of this increased heart action has been explained in several ways, some hold that there is a direct toxic action on the myocardium, while others believe that the toxin acts on the vasomotor nerves, and that the effect on the cardiac musculature is secondary. Cases of this kind are met with so frequently in clinical medicine and have been so frequently described that we shall not discuss this today, but shall briefly mention some less common causes of rapid heart action, after which we shall discuss paroxysmal tachycardia and show patients suffering from this condition.

After certain microbial infections, especially diphtheria and influenza, there may be a definite and persistent tachycardia. This may also ensue after wasting diseases without fever, in tuberculosis, and following typhoid. In these cases the tachycardia is probably due to an increased demand on a weakened myocardium. In severe anemias, whether of the secondary or of the pernicious type, or chlorosis, there is usually an associated

tachycardia Tachycardia may result from changes in the extracardial nerves The vagi may be involved by organic changes such as fibrosis of the mediastinal glands, thoracic aneurysms, or new growths, or the accelerator nerves may be involved The changes may be functional rather than organic, but the tachycardia remains the same The so-called labile pulse of the neurasthenic may be caused by this mechanism, that is, by functional rather than by organic changes In certain diseases of the central nervous system the same nerves may be at fault, but here the change is usually in the nucleus of origin or in the central pathway rather than a local peripheral change

Occasionally tachycardia may result from drugs In certain individuals digitalis, belladonna, and alcohol produce an increase of the rate of the heart instead of a decrease In these cases the tachycardia is usually of short duration and disappears as soon as the drug is eliminated Valvular disease may produce rapid heart action when the muscle is exhausted after working against the handicap of a valvular lesion or when the muscle itself is suffering from the same degenerative lesion as the valve Such hearts may beat with a markedly increased rate even when at rest Myocardial degeneration alone will often produce a tachycardia, thus being particularly true in cases of fatty degeneration or fibrosed hearts

Paroxysmal tachycardia or periodic acceleration of the heart rate is a condition often met with clinically The attacks may be transitory and mild, or this may be a grave malady, irremediable, and occasionally directly fatal It is characterized by an abrupt onset, the patient being conscious of a thump or throb in the precordial region, then a fluttering sensation within the left chest In the mild cases and especially in young individuals there is usually no alarm, and the patient continues with his usual activities This mild form is exemplified by our first case

Case I—Mrs R W, age twenty-seven, housewife The patient was first seen in 1924, and at that time the physical examination was negative The heart was of normal size, no murmurs, rate regular, and 74 per minute There was nothing

of importance in the past or family history. The patient was seen in an attack in October. The attack began abruptly, lasted a few minutes, and ended quickly. She was conscious at this time of the rapid rate, and when asked to observe closely any sensations in the precordium, said there was a slight "fluttering feeling." A paroxysm never interfered with her occupation, but paroxysms were more frequent in the past few months than in the preceding three years. An electrocardiogram taken during an attack showed that the tachycardia was auricular in origin and the rate 175 per minute. The beginning of a paroxysm was registered on two later occasions, and after an extrasystole the rapid rate was maintained for a few minutes, and then the normal slow rate was resumed equally rapidly. The site of origin in the auricle was the same in the two tracings. This patient was given small doses of digitalis, and at the end of a month reported less frequent attacks. She continued to care for her house and 3 children without any trouble. She has discontinued the digitalis at present and still has an occasional paroxysm without any discomfort.

In certain cases with associated valvular, myocardial, or arterial changes the symptoms referable to cardiac insufficiency may be increased during an attack.

This condition is illustrated by our second patient, who was a merchant forty-five years of age and who had had four attacks of rapid heart action in the past two years. In addition to the tachycardia there were also some palpitation and a sensation of precordial pressure, but not of pain. Each attack had followed intense emotional excitement. The attacks previously lasted several hours, but the last one was present for about thirty-six hours. The patient had been able to carry on his ordinary occupation, but was more cautious of physical exertion during an attack. He felt perfectly well between attacks except for some shortness of breath on unusual exertion. There were never dizziness, edema, or dyspnea while at rest. There was nothing of importance in the past or family history.

Physical examination at the time of admission showed a man who was apparently very ill. He appeared very anxious,

was sitting in his chair, and was breathing with considerable difficulty. The skin felt cold and clammy, but there was no edema. The heart was slightly enlarged to the left, the rhythm was regular, and the rate was 160 per minute. The blood-pressure was normal. Large doses of quinidin sulphate were given, 10 grains every four hours, until 60 grains were taken. The attack stopped after the second dose and the patient felt well within half an hour. As this was thirty-six hours after the onset of the attack, the efficacy of the drug was questioned. An electrocardiogram could not be taken at this time, but one was taken a few weeks later, and was perfectly normal. A distance plate of the heart showed normal diameters. The patient had another attack after being sent home, and returned with a second attack in February. The attack had lasted thirty hours at the time of admission to the hospital, and an electrocardiogram at this time showed the tachycardia to be of auricular origin. The rate was 166 per minute and quite regular. A distance plate showed very slight enlargement of the heart after twenty-six hours. Quinidin and digitalis intravenously were given as the patient began to look like one with cardiac decompensation. The patient complained of precordial pressure and as if his heart were in a vise. There was a fluttering sensation in the chest, but no pain. After a few more hours the patient told the nurse that the attack was over, he felt a marked throb, and then the heart seemed to beat very slowly. The pulse at this time was 80 per minute. When seen half an hour later he appeared comfortable, but there was still some edema of the legs. A soft systolic murmur was found at the apex after the attack, a finding which was not present before. There were no symptoms of a thyrotoxic state and the basal metabolism was normal.

The patient had no further attacks for four months, after which he was again attacked with rapid heart action and with signs of complete cardiac decompensation. There were now continual angina-like pains which radiated to the left arm. Physical examination revealed nothing in the heart except a rapid and regular heart action. He received digalin, 3 cc., intra-

venously in twenty-four hours, with no results. The patient grew worse and died forty-six hours after the last attack began.

This is an example of what may occur in such an apparently simple condition as paroxysmal tachycardia. Such results are very rare, but paroxysmal tachycardia is not always the mild and harmless condition we usually consider it to be.

In a given patient the duration of each paroxysm is fairly constant, but the duration in different patients is remarkably variable. One paroxysm may last for six or more beats or it may last for an hour, a day, a week, or even longer without interruption. Very rarely there may be both short and long paroxysms in the same patient, but it is difficult to decide just what constitutes a single paroxysm of tachycardia. Extrasystoles frequently occur in groups, thus two or three or more extrasystoles may occur in regular order, and in these cases there can be no dividing line between these disturbances and the very short paroxysms of tachycardia which last for only a few beats. Frequently these short paroxysms of half a dozen beats occur immediately before the beginning of, or immediately after the end of a long-continued paroxysm. As in this case, it is common to find occasional extrasystoles during the normal beating of the heart, and these beats arise in the same focus as those of the paroxysm of tachycardia.

The focus in which these paroxysms arise is both of interest and of importance. It is possible to determine this point of origin by venous curves, but it is more easily localized by electrocardiographic tracings. Cases in which the paroxysm is auricular in origin usually have a better prognosis than those in which it is ventricular, although this was not true in our case. In interpreting the electrocardiographic tracing it is often difficult to decide if a given paroxysm is of ventricular origin or if it is auricular with aberrant ventricular response.

The treatment of paroxysmal tachycardia is a mooted point. It has been stated that quinidin sulphate stops attacks, but this has not proved true. Digitalis has given good results when the attacks are frequent and of short duration and the effect seems

to be on the ectopic focus, rendering it less irritable, so that the attacks do not begin Nothing seems to be of great value after the attack has begun and symptomatic treatment is the best we have to offer Associated disease of the myocardium or valves requires the chief attention, as the tachycardia may be the result of these abnormal conditions

INDEX TO VOLUME 9

- ABDOMINAL adhesions, angina pectoris secondary to, *March*, 1245
crisis, acute, heart disease and, *Jan*, 988, 1030
disturbances, chronic, heart disease and, *Jan*, 988, 1031
viscera, effects of massage on, *Sept*, 296
- Abscess, Bezold's, *Marcl*, 1301, 1387
of liver following treatment of amebic colitis, *Jan*, 1167
- Accessory nasal sinuses, focal infection in, anoxemia and, *Nov*, 725
- Acidosis and coma, diabetic, *July*, 76
- Acromegaly, chloasma in, case report, *July*, 19
etiological influence of pregnancy in, *July*, 20
hair growth in, *July*, 14
vertex alopecia of, *July*, 8
- Acute indigestion associated with coronary disease, *March*, 1274
yellow atrophy of liver, *May*, 1598
- Addison's disease, pigmentation of, *July*, 3
- Adhesions, abdominal, angina pectoris secondary to, *March*, 1245
- Ainhum, *Jan*, 1181
differential diagnosis, *Jan*, 1184
etiology, *Jan*, 1181
pathology, *Jan*, 1182
prognosis, *Jan*, 1184
symptoms, *Jan*, 1183
treatment, *Jan*, 1184
- Akasia, *Nov*, 629, 635, 637
- Albuminuria, orthostatic, *May*, 1591
- Alcoholic psychosis refractive index of cerebrospinal fluid in, *Jan*, 1126
- Alkali therapy, alkalosis following, *Sept*, 349
- Alkalosis, *Sept*, 343
after persistent vomiting, *Sept*, 353
associated with fever, *Sept*, 354
with intestinal obstruction, *Sept*, 351
chemistry of, *Sept*, 343
following alkali therapy, *Sept*, 349
Sippy treatment, *Sept*, 348
- Alkalosis, ketosis associated with, *Sept*, 354
secondary to persistent vomiting caused by ventral hernia, *Sept*, 347
symptoms, *Sept*, 343
presented by Henoch's type of idiopathic purpura, *Sept*, 345
treatment, *Sept*, 355
- Allotriophagia, *Nov*, 638
- Alopecia, marginal, *July*, 8
of hypothyroidism, *July*, 8
of pituitary disorder, *July*, 8
vertex, *July*, 8
- Amaurosis, fleeting, *Nov*, 813
- Amebic colitis, treatment of, abscess of liver following, *Jan*, 1167
dysentery, *Jan*, 881
chronic, stovarsol in, *Jan*, 1089
stovarsol in, *Jan*, 1167
- Aments, *July*, 184
- Amputation in diabetes, intoxication following, *July*, 169
- Amyotrophic lateral sclerosis, *May*, 1479, 1541
- Anemia, pernicious, case illustrating, *May*, 1568
relation of sprue to, *Jan*, 895, 904
sickle-cell, *Jan*, 1191
pathogenesis, *Jan*, 1196
prognosis, *Jan*, 1197
treatment, *Jan*, 1197
splenic, *May*, 1586
- Banti's disease and, differentiation, *May*, 1589
- Gaucher's disease and, differentiation, *May*, 1589
- Aneurysm, arteriovenous, *Jan*, 1131
intrathoracic growth simulating, *Jan*, 1109
- Angina pectoris, *Marcl*, 1439
associated with coronary disease, *March*, 1268
prognosis, *March*, 1448
secondary to abdominal adhesions, *March*, 1245
symptoms, *Marcl*, 1441
treatment, *Marcl*, 1449

- Anorexia, elective secondary mental, *Sept.*, 337
 primary mental, *Sept.*, 335
 secondary mental, *Sept.*, 336
- Anoxemia, focal infection in nose, throat, accessory nasal sinuses and teeth and, *Nov.*, 725
 in carbon monoxid poisoning, early recognition and treatment, *Sept.*, 473
- Antrum, *March*, 1293
- Appendicitis, chronic, duodenal ulcer and, x-rays in differential diagnosis, *Nov.*, 803
- Appetite, *Nov.*, 629
 abnormal, *Nov.*, 629
 and hunger, differences between, *Nov.*, 630
 disturbances of, as manifestation of gastric neuroses, *Nov.*, 629
 excesses of, *Sept.*, 338
- Arrhythmia in hyperthyroidism, *Nov.*, 659
 quinidin in, *Nov.*, 617
- Arsenical treatment of syphilis, value of liver function tests during, *Nov.*, 613
- Arteriovenous aneurysm, *Jan.*, 1131
- Artery, cerebellar, postero-inferior, occlusion of, *May*, 1544
 pulmonary, anatomy of, *Nov.*, 674
 physiology of, *Nov.*, 676
 sclerosis of, *Nov.*, 673 See also *Sclerosis of pulmonary artery*
- Arthritis, chronic, modern treatment of, *Sept.*, 278
 physical therapy in, *Sept.*, 277
 autocondensation, *Sept.*, 288
 bactericidal effect of ultraviolet rays, *Sept.*, 281
 electricity, *Sept.*, 283
 galvanism, *Sept.*, 284
 heat in, *Sept.*, 282
 practical application, *Sept.*, 283
 heliotherapy, *Sept.*, 280
 high colonic irrigations, *Sept.*, 295
 high-frequency currents, *Sept.*, 286
 hydrotherapy, *Sept.*, 290
 local application, *Sept.*, 294
 methods of applying, *Sept.*, 293
 prescription for, *Sept.*, 294
 in arthritis deformans, *Sept.*, 303
 in degenerative arthritis, *Sept.*, 305
- Arthritis, chronic, physical therapy in gonococcus arthritis, *Sept.*, 305
 in proliferative arthritis, *Sept.*, 300
 limitations of, *Sept.*, 308
 massage, *Sept.*, 295
 mechanotherapy and exercises, *Sept.*, 299
 Oudin current, *Sept.*, 288
 physiologic effects of ultraviolet light, *Sept.*, 281
 practical application of ultraviolet rays, *Sept.*, 282
 prevention of deformity, *Sept.*, 306
 radium and x-ray, *Sept.*, 290
 sanitarium treatment, *Sept.*, 300
 sinusoidal current, *Sept.*, 285
 static machine, *Sept.*, 288
 sugar tolerance tests in, *Nov.*, 559
- Aspiration in pleurisy, *Jan.*, 971
- Asthma, bronchial, pulmonary tuberculosis in early infancy simulating, *Nov.*, 827
 sclerosis of pulmonary artery and, differentiation, *Nov.*, 710
 cardiac, *Jan.*, 1033, 1038
 syphilis of trachea and bronchi simulating, *Sept.*, 359
- Ataxia, Friedreich's, *May*, 1479
- Atresia, congenital, of duodenum, by adhesion, *May*, 1580
- Atrium, *March*, 1290
- Atrophy, acute yellow, of liver, *May*, 1598
 progressive muscular, *May*, 1477
- Attic, *March*, 1290
- Auditory nerve, tumor of cerebello-pontine angle arising from, *May*, 1552
- Auricular fibrillation in hyperthyroidism, *Jan.*, 1097
 nature, *Jan.*, 1200
 quinidin in, *July*, 227, *Nov.*, 617, *Jan.*, 1199 See also *Quinidin in auricular fibrillation*
- Autocondensation in treatment of chronic arthritis, *Sept.*, 288
- Ayerza's disease, *Nov.*, 707
- BABINSKI sign, *May*, 1476
- Bacillary dysentery, chronic, *Jan.*, 1167
- Bacillus, Hansen's, *Jan.*, 1065
- Bacterial endocarditis, cholecystitis induced by, clinical picture, *Sept.*, 325

- Bactericidal effects of ultraviolet rays in chronic arthritis, *Sept.*, 281
 Bacteremia, meningococcus, *Sept.*, 461
 Baldness, hypothyroid, *July*, 8
 localized, and hypothyroidism, *July*, 3
 Banti's disease, splenic anemia and, differentiation, *May*, 1589
 Basal metabolism in hyperthyroidism, *Nov.*, 660
 Belching a gastro-intestinal neurosis, *Sept.*, 340
 Bence-Jones protein in urine in multiple myeloma, *July*, 258
 Bezold's abscess, *March*, 1301, 1387
 Bile, functions of, *Sept.*, 517
 influence of, on digestion and absorption of fat, *Sept.*, 518
 normal secretion and absorption of, *Sept.*, 517
 pigment in blood-serum, van den Bergh test for, *Sept.*, 514
 Bilirubin, *Sept.*, 509
 Bilobal hypopituitarism, case report, *July*, 16
 Binet's tests for mentally defective infant and child, *July*, 61, 62
 Bismuth in visceral syphilis, *Jan.*, 1173
 Bleeding Meckel's diverticulum, *May*, 1585
 Blindness, transitory, after chicken-pox, *Nov.*, 809
 Blood, coagulation of, Howell's theory, *May*, 1567
 diseases of, with special reference to hemorrhages, *May*, 1563
 occult, in stool, importance, *May*, 1601
 Blood pressure in hyperthyroidism, *Nov.*, 659
 Boat-shaped stomach, *Nov.*, 785
 Bone marrow and spleen, metastatic carcinoma of, *July*, 211
 Brain, changes in in syphilis, *July*, 144
 Brain-stem lesions, symptoms, *May*, 1541
 Branham's sign in arteriovenous aneurysm, *Jan.*, 1132, 1137
 Break-bone fever, *Jan.*, 916
 Bromsulphthalein test of liver function, *Jan.*, 1226
 Bronchi and trachea, syphilis of, simulating asthma, *Sept.*, 359
 pulmonary tuberculosis, *Sept.*, 361
 Bronchial asthma, pulmonary tuberculosis in early infancy simulating, *Apr.*, 827
 Bronchial asthma, sclerosis of pulmonary artery and, differentiation, *Nov.*, 710
 Bulimia, *Nov.*, 629
 congenital, *Nov.*, 635
 nature of, *Nov.*, 634
 nocturnal, *Nov.*, 631
 treatment, *Nov.*, 633
 CALCIUM and phosphorus in blood-plasma, increase in, from ultraviolet rays, *Sept.*, 281
 Canine hunger, *Nov.*, 631
 Carbon monoxide poisoning, *Sept.*, 473
 Carcinoma, clinically dormant, *May*, 1627
 gastro-intestinal, sugar tolerance tests in, *Nov.*, 561
 metastatic, of bone-marrow and spleen, *July*, 211
 nasal, *March*, 1381
 primary, of lung, *Jan.*, 887
 Cardiac asthma, *Jan.*, 1033, 1038
 decompensation in hyperthyroidism, *Nov.*, 659
 hemoptysis, *Nov.*, 695
 irregularity, quinidin in, *Nov.*, 617, 628
 murmurs in hyperthyroidism, *Nov.*, 658
 Cardiospasm, *Nov.*, 853
 diagnosis, *Nov.*, 855
 etiology, *Nov.*, 853
 instrumentation in, *Nov.*, 856
 symptoms, *Nov.*, 854
 treatment, *Nov.*, 858
 Cardiovascular renal disease, dietetic management, *July*, 131
 symptoms of hyperthyroidism, *Nov.*, 656
 Cascade stomach, *Nov.*, 769, 800
 Catarrhal jaundice, severe protracted, malignancy and, value of liver function tests in differentiating, *Nov.*, 608
 Cavernous hemangioma of nasal septum, *March*, 1382
 Central vascular system, syphilis of, *Sept.*, 437
 Cells, mastoid, *March*, 1292
 Cerebellar artery, postero-inferior, occlusion of, *May*, 1544
 Cerebellopontine angle, tumor of, arising from auditory nerve, *May*, 1552
 Cerebral hemorrhage, refractive index of cerebrospinal fluid in, *Jan.*, 1126

- Cerebral hemorrhage with intense hyperglycemia and glycosuria, *March*, 1317
- Cerebrospinal fluid, comparative studies, *Jan*, 1115
refractive index, *Jan*, 1115
syphilis, refractive index of cerebro spinal fluid in, *Jan*, 1121
- Chickenpox, transitory blindness after, *Nov*, 809
- Child, mentally defective, *July*, 57
See also *Mentally defective infant and child*
- Childhood, gastro-intestinal hemorrhage in, *May*, 1577
heart disease in, care of, *May*, 1538
infectious mononucleosis in, *Nov*, 589
prevention of heart disease in, *May*, 1531
- Chloasma, *July*, 4
and vitiligo in pituitary disorders, *July*, 20
in acromegaly, case report, *July*, 19
- uternum, *July*, 4
- Cholecystitis induced by bacterial endocarditis, clinical picture, *Sept*, 325
typhoid, *Jan*, 935
- Cholecystoduodenocholic band, duodenal obstruction due to, *March*, 1423
- Chorea, heart disease and, *May*, 1532
- Christie's sign in pericarditis, *Jan*, 1040
- Chromophytosis, *Nov*, 651
- Circulation, effect of massage on, *Sept*, 295
- Cirrhosis of liver, value of liver function tests in, *Nov*, 611
- Cisterna puncture in treatment of hydrocephalus, *July*, 165
- Clinical diagnosis, accuracy in, factor of heredity, *July*, 111
of individual's reactions to his environment, *July*, 113
factors underlying, *July*, 109
picture of cholecystitis induced by bacterial endocarditis, *Sept*, 325
- Clubbed fingers in sclerosis of pulmonary artery, *Nov*, 696
- Coagulation of blood, Howell's theory, *May*, 1567
- Code defect in x ray diagnosis of duodenal ulcer, *Nov*, 795
- Colitis, amebic, treatment of, abscess of liver following, *Jan*, 1167
- Coma and acidosis, diabetic, *July*, 76
diabetic, pitfalls in diagnosis, *Jan*, 959
- Coma, diabetic, without ketonuria, *Jan*, 959
non diabetic, with glycosuria and hyperglycemia, *Jan*, 959
- Common occurrence of renal glycosuria, *Nov*, 549, 569
- Compressive syndrome (pachymeningitis hemorrhagica interna or chronic subdural hemorrhage), *May*, 1558
- Congenital atresia of duodenum by adhesion, *May*, 1580
- Convulsions, infantile, refractive index of cerebrospinal fluid in, *Jan*, 1126
- Coronary disease, *March*, 1261
acute indigestion associated with, *March*, 1274
angina pectoris associated with, *March*, 1268
associated clinical findings, *March*, 1267
coronary T-wave in, *March*, 1261, 1265
diagnosis, *March*, 1277
electrocardiogram in diagnosis, *March*, 1277
heart muscle failure associated with, *March*, 1271
pathology, *March*, 1277
prognosis, *March*, 1280
treatment, *March*, 1280
obstruction, acute, *March*, 1274
T-wave in coronary disease, *March*, 1261, 1265
- Cough in heart disease, *Jan*, 1032
- Counterirritation from ultraviolet rays in nerve pains, *Sept*, 282
- Crisis, acute abdominal, heart disease and, *Jan*, 988, 1030
- Cup and spill stomach, *Nov*, 769
- Cyanosis in sclerosis of pulmonary artery, *Nov*, 696
- DACTYLOLYSIS spontaneous, *Jan*, 1181
- Damages, disability, or disease, *May*, 1499
- Dandy fever, *Jan*, 916
- Death, sudden, following prophylactic inoculation against diphtheria, *March*, 1417
- Decompensation, cardiac, in hyperthyroidism, *Nov*, 659
- Delirium, digitalis, *March*, 1391
- Dementia or mental deficiency, *July*, 183
- Dengue fever, *Jan*, 915
animal transmission, *Jan*, 929
clinical cases, *Jan*, 919-921
etiology, *Jan*, 924
mosquito transmission, *Jan*, 921

- Dengue fever, pathology, *Jan*, 930
symptoms, *Jan*, 916
- Dermatitis repens, *Nov*, 641
- Dermatophytosis, *Nov*, 640
- Deviated nasal septum, formation of, *Nov*, 732
- Dhobie itch, *Nov*, 643
- Diabetes mellitus, amputation in, intoxication following, *July*, 169
and pregnancy, *May*, 1491
associated with pulmonary tuberculosis, management, *May*, 1453
- fusospirochetal pulmonary infection complicating, *March*, 1433
in infants, *July*, 79
and children, *July*, 69
acidosis and coma in, *July*, 76
autogenous carbohydrate tolerance, *July*, 75
effect of prolonged insulin on carbohydrate tolerance, table showing, *July*, 82-84
glucose in acidosis, *July*, 78
glycosuria in, not of pancreatic origin, *July*, 81
insulin requirement, *July*, 72
nutrition and growth in, *July*, 69
protein allotment in, *July*, 70
protocols of representative cases, *July*, 85
Shaffer's formula for carbohydrate allowance, *July*, 71
treatment for acidosis in, *July*, 77
in relation to gastro intestinal disease, *Nov*, 843
treatment, *Nov*, 845
insulin in, dosage, *Jan*, 943
sugar tolerance tests in, *Nov*, 563
- Diabetic acidosis and coma, *July*, 76
coma pitfalls in diagnosis, *Jan*, 959
without ketonuria, *Jan*, 959
- Diagnosis, neurologic, *May*, 1473
- Diaphragmatic hernia, classification, *Jan*, 1075
diagnosis *Jan*, 1077
differential, *Jan*, 1078
etiology, *Jan*, 1076
non traumatic, *Jan*, 1073
case report *Jan*, 1079
prognosis, *Jan*, 1078
situation, *Jan*, 1076
symptoms, *Jan*, 1078
treatment, *Jan*, 1078
- Diathermy in treatment of chronic arthritis, *Sept*, 286
- Diathermy in treatment of chronic arthritis, absorptive or sedative, *Sept*, 287
medical, *Sept*, 287
stimulative, technic, *Sept*, 287
surgical, *Sept*, 287
- Dick test in scarlet fever, *Jan*, 1240
technic, *Jan*, 1242
- Diet in typhoid fever, *Jan*, 939-941
- Dietetic management of cardiovascular renal disease, *July*, 131
acidity of foods, Sansum's table, *July*, 133
alkali-producing foods, Sansum's table, *July*, 134
cases illustrating, *July*, 136-140
diet for ambulant hypertension patient, *July*, 135
salt-free diet in, *July*, 131
Sansum's basic foods, *July*, 135
- Digitalis delirium, *March*, 1391
hypersusceptibility and dosage, *Nov*, 575
cases of hypersusceptibility, *Nov*, 577
dosage and administration, *Nov*, 586
drops, *Nov*, 586
Eggleston's contribution, *Nov*, 575
evidences of digitalization, *Nov*, 577
Pardee's findings, *Nov*, 581
persistence of effect, *Nov*, 582
preparations *Nov*, 583
difficulty in standardization of, *Nov*, 585
rate of disappearance, *Nov*, 581
when to stop digitalis, *Nov*, 588
- Diphtheria, pleural complications of, *July*, 221
prophylactic inoculation against, sudden death following, *March*, 1417
- Disability, damages, or disease, *May*, 1499
- Disease, disability, or damages, *May*, 1499
- Disidrosis, *Nov*, 641
- Diuretin in hydrocephalus, *July*, 48, 51, 52
- Diverticulum, hour-glass, *Nov*, 797
Meckel's bleeding, *May*, 1586
pulsion, *Nov*, 760, 761

- Diverticulum, traction, *Nov*, 761
 Duchez's serum in scarlet fever, *Jan*, 1239
 Dosage of insulin, *Jan*, 943
 Drum membrane, *March*, 1291
 Ductless gland disorder, distribution of hair growth as diagnostic sign, *July*, 4, 8
 Duke's method of determining bleeding time, *March*, 1359
 Duodenal lavage, clinical results with, *July*, 223
 obstruction due to cholecystoduodenal biliary, *March*, 1423
 ulcer, chronic appendicitis and, x-rays in differential diagnosis, *Nov*, 803
 functional diseases of stomach and, x-rays in differential diagnosis, *Nov*, 803
 gall-bladder disease and, x-rays, in differential diagnosis, *Nov*, 803
 hypersensitivity to milk complicating treatment, *March*, 1409
 in child eight and a half years of age, *May*, 1577
 peritoneal adhesions and, x-ray in differential diagnosis, *Nov*, 804
 x-ray diagnosis, *Nov*, 755, 787
 bulbar retraction, *Nov*, 798
 bulbous pouch, *Nov*, 797
 cascade stomach, *Nov*, 800
 Cole defect, *Nov*, 795
 differential, *Nov*, 803
 dilatation of cap, *Nov*, 798
 diverticulum, *Nov*, 797
 en face niche, *Nov*, 792
 gastropasm, *Nov*, 800
 indirect signs, *Nov*, 799
 localized pressure point over ep, *Nov*, 798
 niche, *Nov*, 791
 phthisis bulbi, *Nov*, 799
 profile niche, *Nov*, 792
 teeth like contractions, *Nov*, 800
 Duodenum, anomalies of, *Nov*, 789
 congenital stenosis of, by adhesion, *May*, 1580
 Dye test, value of, in detecting presence of liver metastases before operation, *Nov*, 612
 Dysentery, amebic, *Jan*, 881
 chronic, stovarsol in, *Jan*, 1089, 1167
 bacillary, chronic, *Jan*, 1167
 entamebic, unusual clinical types, *Jan*, 1045-1056
 Dysplasias, *Jan*, 1211
 Dyspnea in sclerosis of pulmonary artery, *Nov*, 697
 Dysrhythmia intermittens, angiosclerotic, pulmonis, *Nov*, 713
 Dystrophy, pseudohypertrophic muscular, *May*, 1478
 Ear cases, demonstration of, *March*, 1375
 middle, anatomy of, *March*, 1288
 Earache in acute otitis media, *March*, 1295
 Eczema in infancy due to protein sensitization, *Nov*, 817
 Eczematized ringworm of extremities, *Nov*, 639
 Edema in chronic nephritis, treatment, *Sept*, 434
 Effleurage, *Sept*, 298
 Effort syndrome, *March*, 1445, 1448
 Electricity in treatment of chronic arthritis, *Sept*, 283
 Electrocardiogram in diagnosis of coronary disease, *March*, 1277
 Electrocardiographic studies, value of, in unusual manifestations of heart disease, *Jan*, 985
 Electrocoagulation in treatment of chronic arthritis, *Sept*, 287
 Embolism, sclerosis of pulmonary artery and, differentiation, *Nov*, 710
 Emphysema, sclerosis of pulmonary artery and, differentiation, *Nov*, 709
 Empyema of unusual origin, *March*, 1413
 Encephalitis lethargica, refractive index of cerebrospinal fluid in, *Jan*, 1124
 Encephalopathy, lead, mimicking an acute abdominal condition, *March*, 1367
 Endocarditis, bacterial, cholecystitis induced by, clinical picture, *Sept*, 325
 symptoms, *Jan*, 1040
 Enface niche in x-ray diagnosis of duodenal ulcer, *Nov*, 792
 Entamoebic dysentery, unusual clinical types, *Jan*, 1045-1056
 Epigastric burning in gastric neurosis, *Sept*, 340
 Epithelioma, sugar tolerance tests in, *Nov*, 562
 Erb's spinal spastic paralysis, *May*, 1477
 Examination of patients with gastrointestinal symptoms, *Sept*, 537

- Exercises in treatment of chronic arthritis, *Sept*, 299
- Exophthalmic goiter of peculiar type, *March*, 1255
- FARADISM in treatment of chronic arthritis, *Sept*, 284
- Fever, alkalosis associated with, *Sept*, 354
- Fibrillation, auricular, in hyperthyroidism, *Jan*, 1097
nature of, *Jan*, 1200
quinidin in, *Jan*, 1199 See also *Quinidin in auricular fibrillation*
- Fingers, clubbed, in sclerosis of pulmonary artery, *Nov*, 696
- Fistula, arteriovenous, *Jan*, 1131
- Flatulence a gastric neurosis, *Sept*, 341
- Fleeting amaurosis, *Nor*, 813
- Fluid, serous, removal of, in pleurisy, *Jan*, 971
- Focal infection, influence of, in nose, throat, accessory nasal sinuses, and teeth, simultaneous anæmia and, *Nor*, 725
- Former fruste types of goiter, *Nor*, 655
- Fragility test, certain applications, *Jan*, 1157
- Friedreich's ataxia, *May*, 1479
- Fusospirochetal pulmonary infection complicating diabetes mellitus, *March*, 1433
- GALL-BLADDER disease, duodenal ulcer and, x-rays in differential diagnosis, *Nor*, 803
- Galvinism in treatment of chronic arthritis, *Sept*, 284
- Gastric neuroses disturbances of appetite or hunger as manifestation, *Nor*, 629
- Gastro-intestinal carcinom, sugar tolerance tests in, *Nor*, 561
disease, diabetes mellitus in relation to *Nor*, 843
treatment, *Nor*, 845
- hemorrhage in children, *May*, 1577
- neuroses, *Sept*, 331
abdominal distention, *Sept*, 341
autonomic nerve control, *Sept*, 331
belching, *Sept*, 340
elective secondary mental anorexia, *Sept*, 337
epigastric burning, *Sept*, 340
excesses of appetite, *Sept*, 338
- Gastro-intestinal neuroses, flatulence, *Sept*, 341
heart-burn, *Sept*, 340
maintaining tonus and posture, *Sept*, 332
motor mechanism affected by reflexes, *Sept*, 333
nausea, *Sept*, 340
pain in, *Sept*, 333
primary mental anorexia, *Sept*, 335
pyrosis, *Sept*, 340
reflexes from emotional centers affecting, *Sept*, 333
secondary mental anorexia, *Sept*, 336
sense of epigastric weight or fulness, *Sept*, 341
sensitivity of tube, *Sept*, 332
- Symptoms, examination of patients with, *Sept*, 537
- Gastropasm in x-ray diagnosis of duodenal ulcer, *Nor*, 800
- Grucher's disease, splenic anæmia and, differentiation, *May*, 1589
- Geophagia, *Nor*, 629, 638
- Glomerular nephritis, acute, *May*, 1593
chronic, *May*, 1595
- Glucose in acidosis of diabetes mellitus, *July*, 78
- Glycogenolysis, *March*, 1321
- Glycosuria in infants and children not of pancreatic origin, *July*, 81
pigment, *March*, 1322, 1323
renal, common occurrence of, *Nor*, 549, 569
- Goiter, classification, *Nor*, 653, 654
exophthalmic, of peculiar type, *March*, 1255
- former fruste types, *Nor*, 655
- heart, *Jan*, 1094
- HAIR, distribution and character as sign of secretory imbalance, *July*, 4, 8
- growth and pigmentation, group classification, *July*, 6
secondary (pituitary), *July*, 13
studies on, *July*, 1
- and suprarenal gland, *July*, 3
- distribution of, diagnostic sign of ductless gland disorder, *July*, 4, 8
- increased, and pituitarism, *July*, 3
- suprarenal cortex disorders and, *July*, 6
- Horseshoe shaped stomach, *Nor*, 785
- Hansen's bacillus, *Jan*, 1065

- Head, traumatic injuries to, refractive index of cerebrospinal fluid in, *Jan*, 1126
 Headache, hunger, *Nov*, 630
 Heartburn a gastric neurosis, *Sept*, 340
 defects, liver disease caused by, *Sept*, 311
 disease, acute abdominal crisis in, *Jan*, 988, 1030
 chorea and, *May*, 1532
 chronic abdominal disturbances in, *Jan*, 988, 1031
 congenital, sclerosis of pulmonary artery and, differentiation, *Nov*, 705
 cough in, *Jan*, 1032
 in childhood, care of, *May*, 1538
 prevention of, in childhood, *May*, 1531
 primary, hyperthyroidism simulating, *March*, 1337
 psychic disturbances and, *Jan*, 1031
 pulmonary symptoms, *Jan*, 1032
 rheumatic, *Sept*, 393
 rheumatism and, *May*, 1536
 tonsillitis and, *May*, 1535
 unusual manifestations, *Jan*, 985
 affirmation of value of electrocardiographic studies, *Jan*, 985
 alleviation of symptoms, *Jan*, 985
 cases illustrating, *Jan*, 988-1029
 classification, *Jan*, 985
 differential diagnosis, *Jan*, 1034
 differentiation, *Jan*, 985
 explanation, *Jan*, 985
 illustration, *Jan*, 985
 treatment, *Jan*, 985, 1040
 goiter, *Jan*, 1094
 in hyperthyroidism, *Jan*, 1093
 etiology, *Jan*, 1094
 physiologic pathology, *Jan*, 1095
 types, *Jan*, 1097
 muscle failure associated with coronary disease, *March*, 1271
 thyroid, *Jan*, 1093
 thyrotoxic, *Jan*, 1094
 Heat in treatment of chronic arthritis, *Sept*, 282
 Heissunger, *Nov*, 631
 Heliotherapy in treatment of chronic arthritis, *Sept*, 280
 Hemangioma, cavernous, of nasal septum, *March*, 1382
 Hemoclastic crisis test of liver function, *Jan*, 1227
 Hemolytic jaundice, *Sept*, 532
 Hemoptysis, cardiac, *Nov*, 695
 from pharynx and tongue, *March*, 1375
 in sclerosis of pulmonary artery, *Nov*, 695
 Hemorrhage, cerebral refractive index of cerebrospinal fluid in, *Jan*, 1126
 with intense hyperglycemia and glycosuria, *March*, 1317
 gastro-intestinal, in children, *May*, 1577
 subdural, chronic, *May*, 1558
 Hemorrhagic chronic nephritis, *Jan*, 909
 diseases, *May*, 1563
 pachymeningitis, internal, *May*, 1558
 purpura, *March*, 1353
 case illustrating, *May*, 1565
 Henoch's type of idiopathic purpura presenting symptoms of alkalosis, *Sept*, 345
 Heredity, factor of, in accuracy in clinical diagnosis, *July*, 111
 in rheumatic heart disease, *Sept*, 395
 Hernia, diaphragmatic, classification, *Jan*, 1075
 diagnosis, *Jan*, 1077
 differential, *Jan*, 1078
 etiology, *Jan*, 1076
 non-traumatic, *Jan*, 1073
 case report, *Jan*, 1079
 prognosis, *Jan*, 1078
 situation, *Jan*, 1076
 symptoms, *Jan*, 1078
 treatment, *Jan*, 1078
 High colonic irrigations in treatment of chronic arthritis, *Sept*, 294
 High-frequency currents in treatment of chronic arthritis, *Sept*, 286
 Hirsutism of virilism, *July*, 3
 Hodgkin's disease, case illustrating, *May*, 1564
 Hookworm disease, *Jan*, 875
 Hour-glass diverticulum, *Nov*, 797
 x-ray diagnosis, *Nov*, 771
 Howell's prothrombin test, *March*, 1362
 theory of blood coagulation, *May*, 1567
 Hunger, *Nov*, 629
 and appetite, differences between, *Nov*, 630
 canine, *Nov*, 631
 disturbances of, as manifestation of gastric neuroses, *Nov*, 629

- Hunger headache, *Nov.*, 630
wolf's *Nov.*, 631
- Hydrocephalus, *July*, 45
acquired, *July*, 46
case reports, *July*, 47
cisterna puncture in treatment of, *July*, 165
congenital, *July*, 45
spina bifida and, relationship between, *July*, 46
diagnosis, *July*, 46
diuretin in, *July*, 48, 51, 52
dye injection in, *July*, 50
etiology, *July*, 45
meningitis and, *July*, 46
obstruction between ventricles and subarachnoid spaces, *July*, 46, 54
surgery in, *July*, 52
treatment, *July*, 54, 55
- Hydrotherapy in treatment of chronic arthritis, *Sept.*, 290
local application, *Sept.*, 294
methods of applying, *Sept.*, 293
prescription for, *Sept.*, 294
- Hyperplasias, *Jan.*, 1211
- Hypersensitivity to milk complicating treatment of duodenal ulcer, *March*, 1409
- Hypertension in hyperthyroidism, *Nov.*, 659
- Hypertensive cardiovascular renal disease, *July*, 263
causes, *July*, 263
disastrous effects of heightened arterial pressure in, *July*, 264
end results of, *July*, 264
lowering of blood-pressure in, *July*, 272
prognosis and treatment, *July*, 263
renal and non renal groups, *July*, 268
- Hyperthyroidism and dominant cardiovascular symptoms without goiter, *Nov.*, 653
arrhythmia in, *Nov.*, 659
auricular fibrillation in, *Jar.*, 1097
basal metabolism in, *Nov.*, 660
blood pressure in, *Nov.*, 659
cardiac decompensation in, *Nov.*, 659
cardiovascular symptoms, *Nov.*, 656
differential diagnosis *Nov.*, 661
forme fruste types, *Nov.*, 655
heart in, *Jan.*, 1093
etiology, *Jan.*, 1094
physiologic pathology, *Jar.*, 1095
types, *Jar.*, 1097

- Hyperthyroidism, hypertension in, *Nov.*, 659
long-standing, with spontaneous subsidence, *Jan.*, 1103
masked as cardiovascular disease with hypertension and arrhythmia, case illustrating, *Nov.*, 662
murmurs in, *Nov.*, 658
simulating primary heart disease, *March*, 1337
sugar tolerance tests in, *Nov.*, 559
symptoms, *Nov.*, 656
tachycardia in, *Nov.*, 658
tinnitus with cardiovascular symptoms, case illustrating, *Nov.*, 666
treatment, *Nov.*, 662
vasomotor disturbances in, *Nov.*, 658
with cardiovascular symptoms in young man, simulating acute endocarditis, *Nov.*, 669
- Hypertrichosis and pigmentation, primary (suprarenal cortex), *July*, 24
of virilism, *July*, 10
secondary, of pituitary disorders, *July*, 13
- Hypertrophic pulmonary osteo-arthropathy, *Nov.*, 696
- Hypogonadism, hypotrichosis of, *July*, 10
- Hypophysis and pigmentation, *July*, 4 and suprarenal gland, similarity of, *July*, 7
- Hypopituitarism, bilobar, case report, *July*, 16
- Hypothyroid baldness, *July*, 8
- Hypothyroidism, *July*, 3
marginal alopecia of, *July*, 9
- Hypotrichosis, *July*, 2
generalized, case report, *July*, 11
of hypogonadism, *July*, 10
universal, *July*, 10
- Hypotympanic space, *March*, 1290
- Hysteresis, refractive index of cerebro-spinal fluid in, *Jan.*, 1124
- Icterus, differential diagnosis, *Sept.*, 509
associated with heart disease, *Sep.*, 533
bilirubin in, *Sept.*, 509
catarrhal, symptoms, *Sept.*, 527
chronic, symptoms, *Sept.*, 528, 531
clinical symptoms according to seat of disease, *Sept.*, 520
dynamic form, symptoms *Sep.*, 525

- Icterus, differential diagnosis, from carcinoma of liver, symptoms, *Sept.*, 531
 functions of bile, *Sept.*, 517
 gravis, symptoms, *Sept.*, 529
 Hanot's, symptoms, *Sept.*, 530
 hemolytic, *Sept.*, 532
 in infectious diseases, symptoms, *Sept.*, 528
 infectious, symptoms, *Sept.*, 527
 manifestations of, *Sept.*, 519
 normal secretion and absorption of bile in, *Sept.*, 517
 obstructive form, symptoms, *Sept.*, 520
 occurring after psychic trauma, *Sept.*, 534
 of transient nature, *Sept.*, 534
 pathologic changes leading to, *Sept.*, 518
 syphilitic, symptoms, *Sept.*, 529
 types, *Sept.*, 509
 urobilin in, *Sept.*, 515
 excretion, *Sept.*, 534
 index, clinical use of, *Nov.*, 603
 value of, in detecting cases of cholecystitis before development of jaundice, *Nov.*, 604
 in determining whether obstructive jaundice has been relieved by operation, *Nov.*, 606
- Idiopathic purpura of Henoch's type presenting symptoms of alkalosis, *Sept.*, 345
- Inanition, Zed reaction in, *July*, 117
 analysis of symptoms, *July*, 118
 definition, *July*, 118
 mucosal contamination, *July*, 119
 pathogenesis, *July*, 119
 symptoms of, *July*, 117
- Incretory imbalance, hair distribution and character as sign, *July*, 4
- Index, icterus See *Icterus index*
 refractive, of cerebrospinal fluid, *Jan.*, 1115
- Indigestion, acute, associated with coronary disease, *March*, 1274
- Infancy, early, pulmonary tuberculosis in, simulating bronchial asthma, *Nov.*, 827
 eczema in, due to protein sensitization, *Nov.*, 817
- Infantile convulsions, refractive index of cerebrospinal fluid in, *Jan.*, 1126
- Infants and children, diabetes melitus in, *July*, 69 See also *Diabetes mellitus*
 glycosuria in, not of pancreatic origin, *July*, 81
 diabetes mellitus in, *July*, 79
 mentally defective, *July*, 57 See also *Mentally defective infant and child*
- Infection, sclerosis of pulmonary artery and differentiation, *Nov.*, 710
- Infection, focal, influence of, in nose, throat, accessory nasal sinuses and teeth, simultaneous anoxemia and, *Nov.*, 725
 mastoid, *March*, 1283
 middle ear, *March*, 1283
- Infectious mononucleosis in childhood, *Nov.*, 589
 incidence, *Nov.*, 593
 resemblance to acute leuke mia, *Nov.*, 592, 596
 typhoid and tuberculosis simulating, *Nov.*, 597
- Influenza meningitis, refractive, index of cerebrospinal fluid in, *Jan.*, 1124
- Insulin, dosage of, *Jan.*, 943
 on day of weekly fast, *Jan.*, 913
 shock accompanying high blood sugar, cell re-education to overcome, *Jan.*, 943
- Intelligence, *July*, 184
 tests, *July*, 185, 186
- Interpretation of sugar tolerance tests, *Nov.*, 549
- Intestinal obstruction, alkalosis associated with, *Sept.*, 351
- Intoxication following amputation in diabetes, *July*, 169
- Intrathoracic growth simulating aneurysm, *Jan.*, 1109
- Iodin metabolism, *March*, 1256
- Itch, dhobie, *Nov.*, 643
 jock strap, *Nov.*, 643
- JAUNDICE See *Icterus*
 Jock strap itch, *Nov.*, 643
- KATAPLASIAS, *Jan.*, 1211
- Kerion ringworm, *Nov.*, 645
- Ketosis associated with conditions of alkalosis, *Sept.*, 354
- LAVAGE, duodenal, clinical results with, *July*, 223
- Lead encephalopathy mimicking an acute abdominal condition, *March*, 1367

- Leather-bottle stomach, *May*, 1609
 Le faim canine, *Nov*, 631
 Leprosy, *Jan*, 1063
 curability, *Jar*, 1071
 epidemiology, *Jan*, 1069
 etiology, *Jan*, 1065
 incubation period, *Jan*, 1070
 Leptomeningitis, syphilitic, tryparsamide in, *Jan*, 981
 Leukemia, case illustrating, *May*, 1570
 etiology, *Jan*, 1212
 nature of, *Jan*, 1207
 treatment of, *Jar*, 1207
 Leukemic states, *Jan*, 1210
 Leukocytosis from ultraviolet rays, *Sept*, 281
 Limitis plastica, *May*, 1609
 Liver, abscess of, following treatment of amebic colitis, *Jan*, 1167
 acute yellow atrophy of, *May*, 1598
 cirrhosis of, value of liver function tests in, *Nov*, 611
 disease caused by heart defects, *Sept*, 311
 pathology in liver, *Sept*, 315
 prognosis in, *Sept*, 321
 symptomatology, *Sept*, 317, 318
 treatment, *Sept*, 321
 types of defects causing, *Sept*, 318
 function, *Jan*, 1219
 tests, *Jan*, 1219
 bromsulphthalein, *Jan*, 1226
 clinical use of, *Nov*, 601
 icterus index, *Nov*, 603
 value of, in detecting cases of cholecystitis, before development of jaundice, *Nov*, 604
 in determining whether obstructive jaundice has been relieved by operation, *Nov*, 606
 Rowntree-Rosenthal test, *Nov*, 602
 value of dye test in detecting presence of liver metastases before operation, *Nov*, 612
 of tests during arsenical treatment for syphilis, *Nov*, 613
 in cases of cirrhosis of liver, *Nov*, 611
- Liver function, tests, clinical use of, value of tests in differentiating between malignancy and severe protracted catarrhal jaundice, *Nov*, 608
 hemoclastic crisis, *Jan*, 1227
 phenol tetra chlorophthalein, *Jan*, 1221
 Rosenthal's, *Jan*, 1219, 1221
 spots, *July*, 4
 Lobar pneumonia, tuberculous, *March*, 1325
 Lung, healed miliary tuberculosis of, with aneurysm of arch of aorta, *Jan*, 1057
 primary carcinoma, *Jan*, 887
 tertiary syphilis of, *Sept*, 364
 Lymphatics, effect of massage on, *Sept*, 296
 Lymphosarcoma, *May*, 1619
 of stomach, *May*, 1615, 1622
 prognosis, *May*, 1624
- MACCALLUM's description of tertiary stage of syphilis, *Sept*, 358
 Macrogenitosomia, *July*, 27
 Malacia, *Nov*, 638
 Malaria, quartan, *Jan*, 863
 Malignancy and severe protracted catarrhal jaundice, value of liver function tests in differentiating, *Nov*, 608
 Marginal alopecia, *July*, 8
 Massage in treatment of chronic arthritis, *Sept*, 295
 contraindications for, *Sept*, 298
 effect in stretching tissue, *Sept*, 296
 on abdominal viscera, *Sept*, 296
 on circulation, *Sept*, 295
 on lymphatics, *Sept*, 296
 mechanical action of, *Sept*, 295
 movements of, *Sept*, 298
 prescription for, *Sept*, 298
 proper use of, *Sept*, 299
 reflex action of, *Sept*, 297
 Massive pericardial adhesions following acute rheumatic fever, *July*, 215
 Mastoid cells, *March*, 1292
 infection, *March*, 1283
 acute, *March*, 1297
 symptoms, *March*, 1298
 treatment, *March*, 1302
 process, anatomy, *March*, 1291
 Mastoiditis, acute, *March*, 1297
 case illustrating, *March*, 1384

- Mastoiditis, acute, double, with septic temperature and followed by measles, *March*, 1385
Bezold's, *March*, 1387
- Mechanotherapy in treatment of chronic arthritis, *Sept.*, 299
- Meckel's diverticulum, bleeding, *May*, 1585
- Mediastinal diseases, sclerosis of pulmonary artery and, differentiation, *Nov.*, 711
- Melanoderma, *July*, 4
- Membrana flaccida, *March*, 1291
 propria, *March*, 1291
- Menigitis, hydrocephalus and, *July*, 46
 influenzal, refractive index of cerebrospinal fluid in, *Jan.*, 1124
 meningococcic, refractive index of cerebrospinal fluid in, *Jan.*, 1124
 tuberculous, refractive index of cerebrospinal fluid in, *Jan.*, 1124
- Meningococcus bacteremia, *Sept.*, 461
 meningitis, refractive index of cerebrospinal fluid in, *Jan.*, 1124
- Mental deficiency or dementia, *July*, 183
- Mentally defective infant and child, *July*, 57
 Binet tests for, *July*, 61, 62
 classification, *July*, 57, 58
 diagnosis, *July*, 59
 family and, *July*, 66
 intelligence of mother a factor in management, *July*, 65
 management of, *July*, 62
 nutrition of, *July*, 65
- Metabolic changes from ultraviolet rays, *Sept.*, 281
- Metabolism, basal, in hyperthyroidism, *Nov.*, 660
 iodin, *March*, 1256
- Metastatic carcinoma of bone-marrow and spleen, *July*, 211
- Middle-ear, anatomy, *March*, 1288
 antrum tenderness, *March*, 1300
 infection, *March*, 1283
- Miliary tuberculosis of lungs, healed, with aneurysm of arch of aorta, *Jan.*, 1057
- Milk, hypersensitivity to, complicating treatment of duodenal ulcer, *March*, 1409
- Monilia psilos, isolation of, *Jan.*, 900
- Mononucleosis, infectious, in childhood, *Nov.*, 589
- Morphea and vitiligo, *July*, 4
 case report, *July*, 23
- Mosquito transmission of dengue fever, *Jan.*, 921
- Multiple myeloma, *July*, 243
 anemia with, *July*, 257
Bence-Jones protein in urine in, *July*, 258
 blood changes in, *July*, 257
 classification, *July*, 256
 differentiation from other osseous involvements, *July*, 260
 etiology, *July*, 253
 nephritis associated with, *July*, 259
 pain in, *July*, 257
 treatment, *July*, 261
 tumor formation in bones in, *July*, 259
- Murmurs in hyperthyroidism, *Nov.*, 658
- Muscular atrophy, progressive, *May*, 1477
 dystrophy, pseudohypertrophic, *May*, 1478
- Myeloma, multiple, *July*, 243
 anemia with, *July*, 257
Bence-Jones protein in urine in, *July*, 258
 blood changes in, *July*, 257
 classification, *July*, 256
 differentiation from other osseous involvements, *July*, 260
 etiology, *July*, 253
 nephritis associated with, *July*, 259
 pain in, *July*, 257
 treatment, *July*, 261
 tumor formation in bones in, *July*, 259
- Myenteric reflex, *Nov.*, 763
- Myocarditis, sclerosis of pulmonary artery and, differentiation, *Nov.*, 703
- Nails, ringworm of, *Nov.*, 649
- Nasal carcinoma, *March*, 1381
 danger zone, *March*, 1288
 septum, cavernous hemangioma of, *March*, 1382
 deviated, formation of, *Nov.*, 732
 importance of, *Nov.*, 729, 730
 sinuses, accessory, focal infection in, anoxemia and, *Nov.*, 725
 syphilis, *March*, 1379
- Nausea a gastro-intestinal neurosis, *Sept.*, 340
- Neoglycogenolysis, *March*, 1323
- Neoplasias, *Jan.*, 1211
- Nephritis, chronic, *Sept.*, 377
 (hemorrhagic), *Jan.*, 909
 treatment, *Sept.*, 427

- Nephritis, chronic, treatment, acidosis in, *Sept.*, 434
 blood letting in, *Sept.*, 435
 cardiac decompensation complicating, *Sept.*, 435
 diet in, *Sept.*, 433
 edema in, *Sept.*, 433, 434
 kidney function in, *Sept.*, 429
 loss of concentrating ability, *Sept.*, 433
 nitrogen retention, *Sept.*, 433
 syndrome, *Sept.*, 432
 sweating in, *Sept.*, 435
 glomerular, acute, *May*, 1593
 chronic, *May*, 1595
 sugar tolerance tests in, *Nov.*, 560
- Nervous run-down patient, *Sept.*, 489
 absence of indicanuria in, *Sept.*, 497
 constipation in, *Sept.*, 496
 treatment, *Sept.*, 497
 constitutional faults of more or less vague nature, *Sept.*, 504
 fibrosis in, *Sept.*, 501
 focal infection in, *Sept.*, 498
 intestinal toxemia in, *Sept.*, 495
 method of studying, *Sept.*, 491
 tonsils in, *Sept.*, 499
 transillumination of sinuses in, *Sept.*, 501
- Neuritis, optic, after chickenpox, *Nov.*, 810
- Neurologic diagnosis, *May*, 1473
- Neuroses, gastric, disturbances of appetite or hunger as manifestation, *Nov.*, 629
- gastro-intestinal, *Sept.*, 331
- Niche in x-ray diagnosis of duodenal ulcer, *Nov.*, 791
 of ulcer of stomach, *Nov.*, 757
- Nocturnal bulimia, *Nov.*, 631
- Non-pancreatic glycosuria in infants and children, *July*, 81
- Nose, carcinoma of, *March*, 1381
 cases, demonstration of, *March*, 1375
 focal infection in, anoxemia and, *Nov.*, 725
 syphilis of, *March*, 1379
- OBSCURE bronchial conditions caused by pollens, *Sept.*, 371
- Occlusion of postero-inferior cerebellar artery, *May*, 1544
- Occult blood in stool, importance, *May*, 1601
- Ochronosis, sclerosis of pulmonary artery and, differentiation, *Nov.*, 712
- Optic neuritis after chickenpox, *Nov.*, 810
- Orthostatic albuminuria, *May*, 1591
- Ossicles, *March*, 1291
- Osteo-arthropathy, hypertrophic pulmonary, *Nov.*, 696
- Otitis media, acute, *March*, 1293
 symptoms, *March*, 1294
 treatment, *March*, 1296
- Oudin current in treatment of chronic arthritis, *Sept.*, 288
- Oxygen, therapeutic use of, in acute respiratory disturbances, *Sept.*, 471
 carbon monoxide poisoning, *Sept.*, 473
 concentration of oxygen in inspired air, *Sept.*, 475
 discussion of results, *Sept.*, 485
 effects of diminished oxygen supply, *Sept.*, 471
 high pressure oxygen tanks, *Sept.*, 475
 illustrative cases, *Sept.*, 479
 methods of giving oxygen, *Sept.*, 475
 nasal catheter method, *Sept.*, 475
 oxygen chambers, *Sept.*, 478
 rebreathing apparatus, *Sept.*, 477
- PACHYMEVAGITIS haemorrhagica interna, *May*, 1558
- Pain in sclerosis of pulmonary artery, *Nov.*, 698
- Paradoxical rest, *Nov.*, 761
- Paralysis agitans, refractive index of cerebrospinal fluid in, *Jan.*, 1124
- Paresis and tryparsamid, method of giving, *July*, 149
 brain changes in, *July*, 144, 146
 clinical diagnosis, *July*, 146
 tryparsamid and, *July*, 143
 case reports, *July*, 150-162
- Paroxysmal tachycardia, *May*, 1632
- Pellagra, *Jan.*, 869
 refractive index of cerebrospinal fluid in, *Jan.*, 1126
- Pericardial adhesions, massive, following acute rheumatic fever, *July*, 215
- Pericarditis, acute rheumatic, diagnosis, *Sept.*, 455

- Pericarditis, Christian's sign, *Jan*, 1040
 sclerosis of pulmonary artery and, differentiation, *Nov*, 704
 symptoms, *Jan*, 1040
 Warthin's sign, *Jan*, 1040
- Peri duodenal adhesions, duodenal ulcer and, x-rays in differential diagnosis, *Nov*, 804
- Pernicious anemia, case illustrating, *May*, 1568
 relation of sprue to, *Jan*, 895, 904
- Petrissage, *Sept*, 298
- Pharynx and tongue, hemoptysis from, *March*, 1375
- Phenoltetrachlorophthalein test of liver function, *Jan*, 1221
- Phosphorus and calcium in blood-plasma, increase in, from ultraviolet rays, *Sept*, 281
- Phthisis bulbi in x-ray diagnosis of duodenal ulcer, *Nov*, 799
- Physical properties of water, *Sept*, 292
 therapy in chronic arthritis its uses and limitations, *Sept*, 277
- Pica, *Nov*, 629
- Pigmentation, absence of, *July*, 4
 and hair growth, group classification, *July*, 6
 secondary, pituitary, *July*, 13
 studies on, *July*, 1
- and hypertrichosis, primary (suprarenal cortex), *July*, 24
 and ultraviolet rays, *Sept*, 281
 in insufficiency of anterior lobe of hypophysis, *July*, 16
 of Addison's disease, *July* 3
- Pigment glycosuria, *March*, 1322, 1323
- Pituitarism and increased growth of hair, *July*, 3
- Pituitary disorders, secondary hypertrichosis of, *July*, 13
 vitiligo and coincident chloasma in, *July*, 20
 gland extract in scleroderma, *July*, 21
 case report, *July*, 23
- Pleural complications of diphtheria, *July*, 221
- Pleurisy, *Jan*, 971
 aspiration in, *Jan*, 971
- Pneumonia, lobar, tuberculous, *March*, 1325
- Pneumothorax, sclerosis of pulmonary artery and, differentiation, *Nov*, 710
- Pollens, obscure bronchial conditions caused by, *Sept*, 371
- Polycythaemia, hypertonic type, case illustrating, *May*, 1574
- Polycythaemia, sclerosis of pulmonary artery and, differentiation, *Nov*, 698
 vera, case illustrating, *May*, 1571
- Polyphagia, *Nov*, 637
- Pompholyx, *Nov*, 641
- Postero inferior cerebellar artery, occlusion of, *May*, 1544
- Postural defects and body types in children, *July*, 99
 treatment, *July*, 106
- Pregnancy, diabetes mellitus and, *May*, 1491
 etiologic influence of, in acromegaly, *July*, 20
- Prepylone line, horizontal, *Nov*, 780
- Profile niche in x-ray diagnosis of duodenal ulcer, *Nov*, 792
- Progressive muscular atrophy, *May*, 1477
- Protective agencies for skin, *Sept*, 291
- Protein, Bence-Jones, in urine in multiple myeloma, *July*, 258
 sensitization, eczema in infancy due to, *Nov*, 817
- Pseudohermaphroditism, *July*, 25, 26, 34
- Pseudohypertrophic muscular dystrophy, *May*, 1478
- Psychic disturbances in heart disease, *Jan*, 1031
- Psychosis, alcoholic, refractive index of cerebrospinal fluid in, *Jan*, 1126
- Pulmonary artery, anatomy, *Nov*, 674
 physiology, *Nov*, 676
 sclerosis of, *Nov*, 673 See also *Sclerosis of pulmonary artery*
- infection, fusospirochetal, complicating diabetes mellitus, *March*, 1433
- osteo arthropathy, hypertrophic, *Nov*, 696
- symptoms of heart disease, *Jan*, 1032
- syphilis, *Sept*, 357
- tuberculosis, diabetes mellitus associated with, management, *May*, 1453
 in early infancy simulating bronchial asthma, *Nov*, 827
 syphilis of trachea and bronchi simulating, *Sept*, 361
- Pulsion diverticulum, *Nov*, 760, 761
- Purpura haemorrhagica, *March*, 1353
 case illustrating, *May*, 1565
- idiopathic, Henoch's type, presenting symptoms of alkalosis, *Sept*, 345
- Purpuras, sclerosis of pulmonary artery and, differentiation, *Nov*, 712

- Pyloric stenosis, hypertrophic congenital, clinical study of 221 operated cases, *March*, 1305
- Pyrosis a gastric neurosis, *Sept*, 340
- QUARTAN malaria, *Jan*, 863
- Quinidin a useful cardiac remedy, *Nov*, 617
- administration, technic, *Nov*, 623
- in arrhythmias, *Nov*, 617
- in auricular fibrillation, *July*, 227, *Nov*, 617, *Jan*, 1199
- action, *Jan*, 1201
- auricular systole in, *July*, 237
- cases illustrating benefits and dangers, *July*, 229-236
- choice of cases, *Nov*, 622
- contraindications, *Jan*, 1202
- dangerous effects, *Jan*, 1205
- dosage, *Jan*, 1204
- effects of restoration of sinus rhythm, *Nov*, 624
- embolism in, *July*, 238
- history, *Jan*, 1199
- indications, *Jan*, 1202
- method of treatment, *Jan*, 1204
- mode of administration, *Jan*, 1204
- proper selection of cases, *Jan*, 1202
- questions on use of, *July*, 228
- rate in, *July*, 236
- relative benefits, *July*, 236
- dangers, *July*, 238
- rhythmicity in, *July*, 237
- routine use, *Jan*, 1202
- toxicity in, *July*, 238
- in cardiac irregularity, *Nov*, 617, 628
- RADIUM and x-ray in treatment of chronic arthritis, *Sept*, 290
- Red and white blood-cells, great increase in in splenomegaly, *July*, 201
- Reflex, myenteric, *Nov*, 763
- Refractive index of cerebrospinal fluid, *Jan*, 1115
- Refractometer, *Jan*, 1119
- Renal glycosuria, common occurrence of, *Aug*, 549, 569
- Respiratory conditions, sclerosis of pulmonary artery and, differentiation, *Nov*, 709
- disturbances, acute, therapeutic use of oxygen in, *Sept*, 471
- Rheumatic fever, acute, massive pericardial adhesions following, *July*, 215
- Rheumatic heart disease, *Sept*, 393
- appearance of signs of cardiac involvement, *Sept*, 397
- cardiac symptomatology, *Sept*, 397
- chronic *Sept* 409
- aortic valvular disease *Sept*, 412
- mitral insufficiency, *Sept*, 409
- stenosis *Sept*, 410
- myocardium in, *Sept*, 415
- pericardium in, *Sept*, 416
- pulmonic insufficiency, *Sept*, 415
- tricuspid valvular disease, *Sept*, 415
- course of, *Sept*, 396
- end-results, *Sept*, 408
- environment in incidence, *Sept*, 395
- etiology, *Sept*, 394
- heredity in, *Sept*, 395
- history, *Sept*, 393
- indications for tonsillectomy in, *Sept*, 422
- pathology, *Sept*, 401
- progress and prognosis of rheumatic carditis, *Sept*, 417
- prophylaxis, *Sept*, 421
- pulse in, *Sept*, 405, 406
- recognition of cardiac infection, *Sept*, 397
- rheumatic nodules in, *Sept*, 398
- symptomatology, *Sept*, 405
- tonsils in, *Sept*, 395
- treatment, *Sept*, 421
- Rheumatism, heart disease and, *May*, 1536
- Ringworm, commoner types, *Nov*, 639
- diagnosis, *Nov*, 639
- eczematized, of extremities, *Nov*, 639
- kerion, *Nov*, 645
- of nails, *Nov*, 649
- of scalp, *Nov*, 644
- treatment, *Nov*, 646
- treatment, *Nov*, 639
- Roentgen ray See x-Ray
- Rosenthal's test of liver function, *Jar*, 1219, 1221
- Rowntree-Rosenthal test, clinical use of, *Nov*, 602
- SCALP, ringworm of, *Nov*, 644
- treatment *Nov*, 646
- Scarlet fever, Dick test in, *Jan*, 1240
- technic, *Jar*, 1242
- etiology, *Jan*, 1233

- Scarlet fever, Schultz-Charlton reaction in, *Jan*, 1236
treatment, *Jan*, 1233
- Schick test on students, *Jan*, 1187
- Schultz-Charlton reaction in scarlet fever, *Jan*, 1236
- Scleroderma, pituitary treatment in, *July*, 21
case report, *July*, 23
- Sclerosis, amyotrophic lateral, *May*, 1479, 1541
of pulmonary artery, *Nov*, 673
anatomy, *Nov*, 674
and its branches, *Nov*, 673
Ayerza's disease and, differentiation, *Nov*, 707
bronchial asthma and, differentiation, *Nov*, 710
clubbed fingers in, *Nov*, 696
congenital heart disease and, differentiation, *Nov*, 705
cyanosis in, *Nov*, 696
diagnosis, *Nov*, 698
dyspnea in, *Nov*, 697
embolism and, differentiation, *Nov*, 710
emphysema and, differentiation, *Nov*, 709
etiology, *Nov*, 677
hemoptysis in, *Nov*, 695
infarction and, differentiation, *Nov*, 710
mediastinal diseases and, differentiation, *Nov*, 711
myocarditis and, differentiation, *Nov*, 703
ochronosis and, differentiation, *Nov*, 712
pain in, *Nov*, 698
pericarditis and, differentiation, *Nov*, 704
physiology, *Nov*, 676
pneumothorax and, differentiation, *Nov*, 710
polycythemia and, differentiation, *Nov*, 698
purpuras and, differentiation, *Nov*, 712
respiratory conditions and, differentiation, *Nov*, 709
symptoms, *Nov*, 694
thrombosis and, differentiation, *Nov*, 710
tuberculosis and, differentiation, *Nov*, 709
- Sensitization, protein, eczema in infancy due to, *Nov*, 817
- Septum, nasal, deviated, formation of, *Nov*, 732
importance of, *Nov*, 729, 730
- Serous fluid, removal of, in pleurisy, *Jan*, 971
- Serum, Dochez's, in scarlet fever, *Jan*, 1239
- Shock, insulin, accompanying high blood-sugar, cell re-education to overcome, *Jan*, 943
- Shrapnell's membrane, *March*, 1291
- Sickle-cell anemia, *Jan*, 1191
pathogenesis, *Jan*, 1196
prognosis, *Jan*, 1197
treatment, *Jan*, 1197
- Sinuses, accessory nasal, focal infection in, anoxemia and, *Nov*, 725
- Sinusitis, chronic, lues plus, *March*, 1377
- Sinusoidal current in treatment of chronic arthritis, *Sept*, 285
- Sippy treatment, alkalosis following, *Sept*, 348
- Skin, protective agencies for, *Sept*, 291
- Snail-form stomach, *Nov*, 768
- Spina bifida, congenital hydrocephalus and, relationship between, *July*, 46
- Spleen and bone-marrow, metastatic carcinoma of, *July*, 211
- Splenic anemia, *May*, 1586
Banti's disease and, differentiation, *May*, 1589
Gaucher's disease and, differentiation, *May*, 1589
- Splenomegaly with great increase in white and red blood-cells, *July*, 201
- Sprue, clinical manifestations, *Jan*, 895
culture of stools, *Jan*, 900
etiology, *Jan*, 902
relation of, to pernicious anemia, *Jan*, 895, 904
symptoms, *Jan*, 898
treatment, *Jan*, 907
- Static machine in treatment of chronic arthritis, *Sept*, 288
- Stenosis, pyloric, hypertrophic congenital, clinical study of 221 operated cases, *March*, 1305
- Stomach, boat-shaped, *Nov*, 785
cascade, *Nov*, 769, 800
cup and spill, *Nov*, 769
functional diseases of, duodenal ulcer and, x-rays in differential diagnosis, *Nov*, 803
half-moon-shaped, *Nov*, 785
hour-glass, x-ray diagnosis, *Nov*, 771
- leather-bottle, *May*, 1609
lesions, complicated, *May*, 1601
multiplicity, *May*, 1601

- Stomach lesions, uncommon, *May*, 1601
 lymphosarcoma of, *May*, 1615, 1622
 snail-form, *Nov*, 768
 syphilis of, *Nov*, 717
 diagnosis, *Nov*, 719
 pathology, *Nov*, 718
 prognosis, *Nov*, 720
 symptoms, *Nov*, 719
 treatment, *Nov*, 721
 tobacco-pouch, *Nov*, 768
 ulcer of, x-ray diagnosis, *Nov*, 755,
 757
 callous ulcer of pylorus,
 Nov, 783
 lesser curvature, *Nov*, 773
 motility disturbance on lesser
 curvature, *Nov*, 776
 niche, *Nov*, 757
 ulcer in pars pylorica and
 pylorus, *Nov*, 777
 of pylorus, *Nov*, 782
 visualization of spasm of
 sphincter pylori, *Nov*, 781
- Stool, occult blood in, importance, *May*, 1601
- Stovarsol in amebic dysentery, *Jan*, 1089, 1167
 in quartan malaria, *Jan*, 866
- Stretching of tissues, massage in, *Sept*, 296
- Subdural hemorrhage, chronic, *May*, 1558
- Sugar tolerance tests, interpretation
 of, *Nov*, 549
 duration of blood-sugar
 curve, *Nov*, 554
 fasting blood-sugar level, *Nov*, 551
 in arthritides, *Nov*, 559
 in diabetic mellitus, *Nov*, 563
 in epithelioma, *Nov*, 562
 in gastro-intestinal carcinoma, *Nov*, 561
 in hyperthyroidism, *Nov*, 559
 in nephritis, *Nov*, 560
 in thrombo angitis obliterans, *Nov*, 561
 maximal value of blood-
 sugar after ingestion of 100
 gm of glucose, *Nov*, 551
 method of procedure, *Nov*, 549
 types of blood sugar curves, *Nov*, 555
- Suprarenal cortex, disorders of, *July*, 25
 abnormal development of genital system, *July*, 31
- Suprarenal cortex, disorders of, classification, *July*, 25
 hair growth and, *July*, 6
 pathology, *July*, 40
 postadolescent type, *July*, 29
 preadolescent type, *July*, 25
 gland and hair growth, *July*, 3
 and hypophysis, similarity of, *July*, 7
- Syndrome, effort, *March*, 1445, 1448
- Weber's, *May*, 1485
- Syphilis, arsenical treatment for, value of liver function tests during, *Nov*, 613
 cerebrospinal refractive index of cerebrospinal fluid in, *Jan*, 1121
 changes in brain in, *July*, 144
 nasal, *March* 1379
 of central vascular system, *Sept*, 437
 aneurysm in, *Sept*, 450, 451
 case reports, *Sept*, 437-447
 enlarged heart in, *Sept*, 450
 pathology, *Sept*, 447
 prognosis, *Sept*, 454
 signs, *Sept*, 449
 symptoms, *Sept*, 448
 treatment, *Sept*, 452
 for syphilis, *Sept*, 452
 of heart, *Sept*, 452
 Wassermann reaction in, *Sept*, 451
 of stomach, *Nov*, 717
 diagnosis, *Nov*, 719
 pathology, *Nov*, 718
 prognosis, *Nov*, 720
 symptoms, *Nov*, 719
 treatment, *Nov*, 721
- of trachea and bronchi simulating asthma, *Sept*, 359
 pulmonary tuberculosis, *Sept*, 361
 plus chronic sinusitis, *March*, 1377
- pulmonary, *Sept*, 357
 refractive index of cerebrospinal fluid in, *Jan*, 1121
 tertiary, MacCallum's description, *Sept*, 358
 of lung, *Sept*, 364
- visceral, bismuth in, *Jan*, 1173
- Syphilitic leptomeningitis, tryparsamide in, *Jan*, 981
- Spina, *May*, 1482
- TABES dorsalis, *May*, 1481
- Tachycardia, *May*, 1630
 in hyperthyroidism, *Nov*, 658
 in thyroid disease, *Jan*, 1097
- paroxysmal, *May*, 1632

- Tachycardia, simple, *May*, 1631
 Tadpoles, dermal pigment of, experiments on, *July*, 5
 Tapotement, *Sept*, 298
 Teeth, focal infection in, anoxemia and, *Nov*, 725
 Tertiary syphilis of lung, *Sept*, 364
 Test, bromsulphthalein, of liver function, *Jan*, 1226
 Dick, in scarlet fever, *Jan*, 1240
 technic, *Jan*, 1242
 for liver function, *Jan*, 1219
 fragility, certain applications, *Jan*, 1157
 hemoclastic crisis, of liver function, *Jan*, 1227
 phenoltetrachlorphthalein, *Jan*, 1221
 Schick, on students, *Jan*, 1187
 van den Bergh, certain applications, *Jan*, 1157
 for bile pigment in blood serum, *Sept*, 514
 Widal's, of liver function, *Jan*, 1227
 Therapeutic use of oxygen in acute respiratory disturbances, *Sept*, 471
 Throat cases, demonstration of, *March*, 1375
 focal infection in, anoxemia and, *Nov*, 725
 Thrombo-angitis obliterans, sugar tolerance tests in, *Nov*, 561
 Thrombosis, sclerosis of pulmonary artery and, differentiation, *Nov*, 710
 Thyrocardiacs, *Nov*, 656
 Thyroid hearts, *Jan*, 1093
 Thyrotoxic heart, *Jan*, 1094
 Tinea circinata, *Nov*, 648
 cruris, *Nov*, 642
 versicolor, *Nov*, 652
 Tinnitus with cardiovascular symptoms of hyperthyroidism, case illustrating, *Nov*, 666
 Tobacco-pouch stomach, *Nov*, 768
 Tongue and pharynx, hemoptysis from, *March*, 1375
 tuberculosis of, *Jan*, 1139 See also *Tuberculosis of tongue*
 Tonsillitis, heart disease and, *May*, 1535
 Trachea and bronchi, syphilis of, simulating asthma, *Sept*, 359
 pulmonary tuberculosis, *Sept*, 361
 Traction diverticulum, *Nov*, 761
 Transitory blindness after chickenpox, *Nov*, 809
 Tryparsamid and paresis, *July*, 143
 Tryparsamid and paresis, case reports, *July*, 150-162
 method of giving, *July*, 149
 in syphilitic leptomeningitis, *Jan*, 981
 Tuberculosis, miliary, of lungs, healed, with aneurysm of arch of aorta, *Jan*, 1057
 of tongue, *Jan*, 1139
 case histories, *Jan*, 1144
 case reports, *Jan*, 1150
 diagnosis, *Jan*, 1142
 etiology, *Jan*, 1140
 historical note, *Jan*, 1139
 incidence, *Jan*, 1142
 pathology, *Jan*, 1140
 prognosis, *Jan*, 1143
 symptoms, *Jan*, 1142
 treatment, *Jan*, 1143
 pulmonary, diabetes mellitus associated with, management, *May*, 1453
 in early infancy, simulating bronchial asthma, *Nov*, 827
 syphilis of trachea and bronchi simulating, *Sept*, 361
 sclerosis of pulmonary artery and, differentiation, *Nov*, 709
 Tuberculous lobar pneumonia, *March*, 1325
 meningitis, refractive index of cerebrospinal fluid in, *Jan*, 1124
 Tumor of cerebellopontine angle arising from auditory nerve, *May*, 1552
 Tympanic cavity, anatomy, *March*, 1288
 space, anatomy, *March*, 1289, 1290
 Tympanum, anatomy, *March*, 1288
 Typhoid cholecystitis, *Jan*, 935
 fever, diet in, *Jan*, 939-941
 pathology, *Jan*, 938
 ULCER, duodenal, hypersensitivity to milk complicating treatment, *March*, 1409
 in child eight and a half years of age, *May*, 1577
 x-ray diagnosis, *Nov*, 755, 787
 See also *Duodenal ulcer, x-ray diagnosis*
 of stomach, x-ray diagnosis, *Nov*, 755, 757
 callous ulcer of pylorus, *Nov*, 783
 lesser curvature, *Nov*, 773
 motility disturbance on lesser curvature, *Nov*, 776
 niche, *Nov*, 757

- Ulcer of stomach, *x-ray diagnosis, ulcer in pars pylorica and pylorus, Nov., 777*
of pylorus, Nov., 782
visualization of spasm of sphincter pylori, Nov., 781
- Ultraviolet rays in chronic arthritis, physiologic effects of, *Sept., 281*
- Uremia, *March, 1399*
- Urobilin, *Sept., 515*
- VAN DEN BERGH test, certain applications, *Jan., 1157*
for bile pigment in blood-serum, Sept., 514
- Varicella, transitory blindness after, *Nov., 809*
- Vascular system, central, syphilis of, *Sept., 437*
- Vasomotor disturbances in hyperthyroidism, *Nov., 658*
- Vertex alopecia, *July, 8*
- Vibration, *Sept., 298*
- Virilism, hirsutism of, *July, 3*
hypertrichosis of, July, 10
- Visceral syphilis, bismuth in, *Jan., 1173*
- Vitiligo and coincident chloasma, *July, 20*
and morphea, July, 4
- Vomiting, persistent, alkalosis after *Sept., 353*
- Water, physical properties of, *Sept., 292*
- Weber's syndrome, *May, 1485*
- White and red blood-cells, great increase in, in splenomegaly, *July, 201*
- Whitfield's ointment in ringworm, *Nov., 642*
- Widal's hemoclastic crisis test of liver function, *Jan., 1227*
- Wolf's hunger, *Nov., 631*
- x-RAY and radium in treatment of chronic arthritis, Sept., 290*
diagnosis of duodenal ulcer, Nov., 755, 787 See also *Duodenal ulcer, x-ray diagnosis*
of hour glass stomach, Nov., 771
of ulcer of stomach, Nov., 755, 757
callous ulcer of pylorus, Nov., 783
lesser curvature, Nov., 773
motility disturbance on lesser curvature, Nov., 776
niche, Nov., 757
ulcer in pars pylorica and pylorus, Nov., 777
of pylorus, Nov., 782
visualization of spasm of sphincter pylori, Nov., 781
- in treatment of ringworm of scalp, Nov., 646*
- WARTHIN's sign in pericarditis, *Jan., 1040*
- ZED reaction in inanition, *July, 117*
- Zoster circumflexus, *Jan., 955*



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CONTENTS OF VOLUME 9

July, 1925

ST. LOUIS NUMBER

	PAGE
Clinic of Dr William Engelbach <i>Missouri's Earliest Sarcomas</i> STUDIES ON HAIR GROWTH AND PIGMENTATION	1
Clinic of Dr McKim Marriott <i>From the Department of Pediatrics Washington University School of Medicine and the St Louis Children's Hospital</i> HYDROCEPHALUS	45
Clinic of Dr Borden S Veeder <i>Washington University School of Medicine</i> THE MENTALLY DEFECTIVE INFANT AND CHILD	57
Clinic of Dr Alexis F Hartmann <i>From the Department of Pediatrics Washington University School of Medicine and the St Louis Children's Hospital</i> DIABETES MELLITUS IN INFANTS AND CHILDREN	69
Clinic of Dr Hugh McCulloch <i>Washington University School of Medicine and the St Louis Children's Hospital</i> POSTURAL DEFECTS AND BODY TYPES IN CHILDREN	99
Clinic of Dr William W Graves, <i>S. Louis University School of Medicine</i> SOME OF THE FACTORS UNDERLYING ACCURACY IN CLINICAL DIAGNOSIS	109
Clinic of Dr John Zahorsky <i>Bethesda Hospital</i> THE ZED REACTION IN INCINERATION	117
Clinic of Dr H W Soper, <i>St Luke's Hospital</i> THE DIETETIC MANAGEMENT OF CARDIOVASCULAR RENAL DISEASE	131
Clinic of Dr Sidney I Schwab <i>Barres Hospital Washington University</i> TEYPASAMID AND PARESIS	143
Clinic of Dr Jules M Brady <i>S. Ann's Hospital</i> CISTerna PUNCTURE IN THE TREATMENT OF HYDROCEPHALUS	165
Metabolism Clinic of Dr William H Olmsted <i>Barres Hospital</i> INTOXICATION FOLLOWING AMPUTATION IN DIABETES	169
Clinic of Dr Francis M Barnes Jr <i>From the Department of Nervous and Mental Diseases of the St Louis University School of Medicine and the Department of Neurosurgery of St Mary's Hospital Senior Class Clinic in Psychiatry</i> MENTAL DEFICIENCY OR DEMENTIA	183
Clinic of Dr Louis H Hempelmann <i>Washington University Medical School</i> A CASE OF STYLONOECAE ASSOCIATED IN ITS LATER STAGES WITH GREAT INCREASE IN BOTH WHITE AND RED BLOOD-CELLS	201
Clinic of Dr J Curtis Lyter <i>S. Anne's Hospital</i> METASTATIC CARCINOMA OF THE LONG BONE MARROW AND SPLEEN MASSEY EXTRACARDIAC LESIONS FOLLOWING ACUTE RHEUMATIC FEVER THE PLEURAL COMPLICATIONS OF DIPHTHERIA	211 215 221
Clinic of Dr Walter Baumgarten <i>S. Luke's Hospital</i> CERTAIN CLINICAL RESULTS WITH DUODENAL LIGATION	223
Clinic of Dr Drew Luten <i>Barres Hospital</i> ON THE USE OF QUINIDINE IN ATRIOVENTRICULAR FIBRILLATION	227
Clinic of Dr Alphonse McMahon <i>Formerly Assistant Physician S. John's Hospital</i> MULTIPLE VISCERAL	243
Contribution by Dr Elsworth S Smith <i>Washington University School of Medicine</i> FOUNDATIONS AND TREATMENT OF HYPERTENSIVE CARDIOVASCULAR RENAL DISEASE	263

September, 1925

NEW YORK NUMBER

	PAGE
Contribution by Drs. Russell L. Clegg and K. G. Hansson, Bellevue Hospital and Hospital for Ruptured and Crippled	
PHYSICAL THERAPY IN CHRONIC ARTHRITIS ITS USES AND LIMITATIONS	277
Clinical Lecture by Dr. Harlow Brooks, City Hospital	
LIVER DISEASE CAUSED BY THIOL DERIVATIVES	311
Clinic of Dr. Nellie H. Foster, New York Hospital	
THE CLINICAL PICTURE OF CHOLECYSTITIS INDUCED BY BACTERIAL ENDOCARDITIS	315
Clinic of Dr. Arthur E. Holland, New York Hospital	
GASTRO-INTESTINAL NEUROSES	331
Clinic of Dr. Arthur E. Choice, New York Post Graduate Hospital	
ALKALOIDS	343
Clinic of Dr. George G. Ornstein Columbia University	
PULMONARY SYMPTOMS OBSCURE BRONCHIAL CONDITIONS CAUSED BY POLLUTION	357
371	
Clinic of Dr. Malcolm Goodridge, Bellevue Hospital	
CHRONIC NEURITIS	377
Clinic of Dr. Ernest P. Burns, Montefiore Hospital for Chronic Diseases	
RHEUMATIC HEART DISEASE	393
Clinic of Dr. David W. Atchley, Presbyterian Hospital	
THE TREATMENT OF CHRONIC NEURITIS	427
Clinic of Dr. Walter P. Anderton, Presbyterian Hospital	
SYMPTOMS OF THE CENTRAL VASCULAR SYSTEM	437
Clinic of Dr. Robert L. Koch, Presbyterian Hospital	
THE DIAGNOSIS OF ACUTE RENAL TUBULAR DISTURBANCES	455
Clinic of Dr. Arthur I. Neergaard, Presbyterian Hospital	
MININOOGEOUS HACERITIS	461
Clinic of Dr. Alvan L. Burroughs, Presbyterian Hospital	
THE INTRAVASCULAR USE OF OXYGEN IN ACUTE RENAL TUBULAR DISTURBANCES	471
Clinic of Dr. Arthur I. Neergaard, Bellevue Hospital	
THE NERVOUS, RUN DOWN PATIENT	489
Contribution by Dr. I. W. Heldt, Beth Israel Hospital	
TERATOLOGY AND ITS DIFFERENTIAL DIAGNOSIS	509
Clinic of Dr. H. H. Fellows, Gastro-Intestinal Department Cornell University Medical College, Assisted by Drs. Webster Russell and Miss Frances Lyons, Clinic Executive	
EXAMINATION OF PATIENTS WITH GASTRO-INTESTINAL SYMPTOMS	537

November, 1925

NEW YORK NUMBER

	PAGE
Contribution by Dr. Herman O. Mosenthal, New York Post-Graduate Medical School and Hospital	
THE INTERPRETATION OF SUGAR TOLERANCE TESTS—THE COMMON OCCURRENCE OF RENAL GLUCOSURIA	549
Clinic of Dr. Walter A. Hustedt, St. Luke's Hospital	
DIABETES HYPERGLUCOSURIA AND DIABETES DOSAGE	575
Clinic of Drs. Murray Harold Bass and Harold Hermon, Mt. Sinai Hospital	
INFECTIOUS MONONEURITIS IN CHILDHOOD	589

CONTENTS OF VOLUME 9

5

	PAGE
Clinic of Dr Howard F Shattuck, Post-Graduate Hospital CLINICAL USE OF LIVER FUNCTION TESTS THE ICERT'S INDEX AND ROWNTREE ROSENTHAL SERUM DYE TEST	601
Clinic of Dr Robert L Levy Presbyterian Hospital QUINIDINE—A USEFUL CARDIAC REMEDY	617
Clinic of Dr B B Crohn Mt Sinai Hospital ULCER	629
Contribution by Dr A Benson Cannon From the Department of Dermatology and Syphilology College of Physicians and Surgeons Columbia University New York DIAGNOSIS AND TREATMENT OF THE COMMON TYPES OF RINGWORM	639
Clinic of Dr A S Blumgarten Lenox Hill Hospital HYPERTHYROIDISM AND DOMINANT CARDIOVASCULAR SYMPTOMS WITHOUT GOITER	653
Clinic of Dr Hyman R Miller, Post-Graduate and Manhatten Hospitals SCLEROSIS OF THE PULMONARY ARTERIA AND ITS BRANCHES	673
Clinic of Dr Joseph M Marcus Mount Sinai Hospital SYPHILIS OF THE STOMACH	717
Clinic of Dr Charles Clinck Passaic Eye Ear Nose and Throat Private Hospital THE INFLUENCE OF FOCAL INFECTION IN THE NOSE THROAT ACCESSORY NASAL SINUSES AND TEETH AND THE SIMULTANEOUS ANEMIA OF INTERNAL MEDICINE. SOME ILLUSTRAATIVE CASES	725
Contribution by Drs I W Held and Irving Gray PRESENT STATUS OF THE DIAGNOSIS OF GASTRIC AND DUODENAL ULCER.	755
Contribution by Dr Bret Ratner New York TRANSTORI BLINDNESS AFTER CHICKENPOX. ECZEMA IN INFANCY DUE TO PROTEIN SENSITIZATION PULMONARY TUBERCULOSIS IN EARLY INFANCY SIMULATING BRONCHIAL ASTHMA	809 817 827
Clinic of Dr Seymour Fiske Midtown Hospital DIABETES MELLITUS IN RELATION TO GASTRO-INTESTINAL DISEASE	843
Clinic of Dr Joseph M Marcus Mount Sinai Hospital CARDIOPATHY	853

January, 1926

TULANE UNIVERSITY NUMBER

	PAGE
Clinic of Dr C C Bass Tulane University QUARTAN MALARIA LICHEN HOOKWORM DISEASE AMERIC DISEAS	863 869 875 881
Clinic of Dr George S Bell Charity Hospital PELVIC CAECOMA OF THE LUNG	887
Clinic of Dr John H Musser Charity Hospital CLINICAL MANIFESTATIONS OF SPUR AND RELATION OF THE DISEASE TO PERNICIOUS ANEMIA	895
Clinic of Dr L. R. DeBuys Tulane Infirmary A CHRONIC UVEITIS (HEMORRHAGIC)	909
Clinic of Drs Charles W Duval and William H Harris Presbyterian Hospital DENGUE FEVER, WITH SPECIAL CONSIDERATION OF THE ETIOLOGY AND TRANSMISSION	915
Clinic of Dr Allan Eustis Charity Hospital THYROID CHOLECYSTITIS	935
Clinic of Dr J Birney Guthrie Charity Hospital DOCTOR OF INSULIN AND TABLE FOR ITS USE. INSULE ON DAY OF WEEKLY FAST. CELL RE-EJECTION TO OVERCOME INSULIN SHOCK ACCOMPANYING HIGH BLOOD-SUGAR	943
Clinic of Dr I F Lehmann Tulane Infirmary PITFALLS IN THE DIAGNOSIS OF DIABETIC COMPLICATIONS	959

Clinic of Dr O W Bethea, <i>Charity Hospital</i> PLEURISY REMOVAL OF SEROUS FLUID	PAGE 971
Clinic of Dr Henry Daspit, <i>City Hospital for Mental Diseases</i> A CASE OF SYPHILITIC LEPTOMENINGITIS TREATED WITH TRYPARSAMID	981
Clinic of Dr George R Herrmann, <i>Charity Hospital</i> THE UNUSUAL MANIFESTATIONS OF HEART DISEASE	985
Clinic of Dr Sidney K Simon, <i>Touro Infirmary</i> SOME UNUSUAL CLINICAL TYPES OF ENTAMERIC DYSENTERY	1045
Clinic of Dr Chaille Jamison, <i>Charity Hospital</i> HEALED MILITARY TUBERCULOSIS OF THE LUNGS WITH ANEURYSM OF THE ARCH OF THE AORTA	1057
Clinic of Dr Ralph Hopkins <i>Charity Hospital</i> THE STATUS OF SOME PROBLEMS IN LEPROSY	1063
Clinic of Dr Leon J Menville <i>Presbyterian Hospital</i> THE MEDICAL ASPECT OF NON-TRAUMATIC DIAPHRAGMATIC HERNIA REPORT OF A CASE SITUATED ON THE RIGHT SIDE, ANTERIORLY	1073
Clinic of Dr Foster M Johns <i>Tulane University</i> THE TREATMENT OF CHRONIC AMEBIC DYSENTERY WITH STOVARSON	1089
Clinic of Dr V J Thacker <i>Charity Hospital</i> THE HEART IN HYPERTHYROIDISM	1093
Clinic of Dr C L Eshleman <i>Touro Infirmary</i> LONG-STANDING HYPERTHYROIDISM WITH SPONTANEOUS SUBSIDENCE AN INTRATHORACIC GROWTH SIMULATING ANEURYSM	1103 1109
Clinic of Dr Hamilton P Jones <i>Charity Hospital</i> COMPARATIVE STUDIES OF CEREBROSPINAL FLUIDS ESPECIALLY IN REFERENCE TO REFRACTIVE INDEX	1115
Clinic of Dr Sam Hobson, <i>Charity Hospital</i> ARTERIOVENOUS ANEURYSM	1131
Clinic of Dr Elizabeth Bass <i>Tulane University</i> TUBERCULOSIS OF THE TONGUE	1139
Clinic of Drs Talbot A Tumbleson and John L Carmichael, <i>Charity Hospital</i> CERTAIN APPLICATIONS OF THE VAN DEN BERGH AND FRAGILITY TESTS	1157
Clinic of Dr Daniel N Silverman, <i>Touro Infirmary</i> CHRONIC BACILLARY DYSENTERY LIVER ABSCESS FOLLOWING TREATMENT OF AMEBIC COLITIS SOME NOTES ON THE USE OF STOVARSON IN AMEBIC DYSENTERY	1167
Clinic of Dr J Holmes Smith, Jr., <i>Charity Hospital</i> BISMUTH IN THE TREATMENT OF VISCERAL SYPHILIS	1173
Clinic of Dr H W Butler <i>Charity Hospital</i> AINHUM (DACTYLOLYYSIS SPONTANEA) THE SCHICK TEST ON STUDENTS	1181 1187
Clinic of Dr Earl Z Browne <i>Charity Hospital</i> SICKLE-CELL ANEMIA	1191
Clinic of Dr Morris J Duffy, <i>Charity Hospital</i> QUINIDINE IN THE TREATMENT OF AURICULAR FIBRILLATION	1199
Clinic of Dr Morell W Miller <i>Charity Hospital</i> THE NATURE AND TREATMENT OF THE LEUKEMIAS	1207
Clinic of Dr D L Hagood <i>Charity Hospital</i> LIVER FUNCTION AND LIVER FUNCTION TESTING	1219
Clinic of Dr Robert E Bratton <i>Charity Hospital</i> ETIOLOGY AND TREATMENT OF SCARLET FEVER	1233

CONTENTS OF VOLUME 9

7

March, 1926

CHICAGO NUMBER

Clinic of Dr Charles Louis Mix <i>Mercy Hospital</i>	PAGE
I. ANGINA PECTORIS SECONDARY TO ABDOMINAL ADHESIONS	1245
II. AN EXOPHTHALMIC GOITER OF PECULIAR TYPE	1255
Clinic of Dr Walter W Hamburger <i>Michael Reese Hospital</i>	
DISEASE OF THE CORONARY VESSELS ANGINA PECTORIS, AND "ACUTE INDIGESTION" (WITH SPECIAL REFERENCE TO THE CORONARY T-WAVE)	1261
Clinic of Dr Richard J Tivnen <i>Mercy Hospital</i>	
MIDDLE EAR AND MASTOID INFECTIONS	1283
Clinic of Drs. Isaac A Abt and Alfred A Strauss <i>From the Pediatric Service of Sarah Morris Hospital for Children and Chicago Lying-in Hospital</i>	
CLINICAL STUDY OF 221 OPERATED CASES OF HYPERSTROPHIC CONGENITAL PYLORIC STENOSIS	1305
Clinic of Dr Arthur R Elliott <i>St Luke's Hospital</i>	
CASE I CEREBRAL HEMORRHAGE WITH INTENSE HYPERGLYCEMIA AND GLYCOSURIA	1317
CASE II TUBERCULOUS LUNG PNEUMONIA	1325
Clinic of Dr Walter S Priest <i>Michael Reese and Chicago Memorial Hospitals</i>	
CASES OF HYPERTHYROIDISM SIMULATING PRIMARY HEART DISEASE	1337
Clinic of Dr Charles Spencer Williamson, <i>Research Hospital University of Illinois</i>	
I A CASE OF PURPURA HELORRHAGICA—FULL DISCUSSION OF BLOOD FINDINGS AND CLINICAL FEATURES	1353
II A CASE OF LEAD ENCEPHALOPATHY UNMASKING AN ACUTE ABDOMINAL CONDITION	1367
Clinic of Dr Robert Sonnenschein <i>Post-Graduate Hospital</i>	
A DEMONSTRATION OF EAR, NOSE AND THROAT CASES	1375
Clinic of Dr James G Carr, <i>Cook County Hospital</i>	
DIGITALIS DELIRIUM	1391
A CASE OF UREMIA	1399
HYPERSENSITIVENESS TO MILK COMPLICATING THE TREATMENT OF DUODENAL ULCER	1409
Clinic of Dr Jesse R Gerstley <i>Northwestern University Medical School and Michael Reese Hospital</i>	
EMPHYEMA OF UNUSUAL ORIGIN	1413
SUDDEN DEATH FOLLOWING PROPHYLACTIC INOCULATIONS AGAINST DIPHTHERIA	1417
Clinic of Dr George O Solem <i>Washington Boulevard Hospital</i>	
DROPOUT OBSTRUCTION DUE TO CHOLECYSTOSTOENTERIC BAND	1423
Clinic of Dr I Pilot <i>Michael Reese Hospital</i>	
FECOSPIROCHETAL PULMONARY INFECTION COMPLICATING DIABETES MELLITUS	1433
Clinic of Dr A C Gilbert <i>St Luke's Hospital</i>	
ANGINA PECTORIS	1439

May, 1926

CHICAGO NUMBER

PAGE

Clinic of Drs. Charles A Elliott and Walter H Nadler <i>Wesley Memorial Hospital</i> <i>Northwestern University Medical School Chicago</i>	PAGE
THE MANAGEMENT OF DIABETES MELLITUS ASSOCIATED WITH PULMONARY TUBERCULOSIS	1453
Clinic of Dr Lewis J Pollock <i>Northwestern University Medical School</i>	
BACTERIOLOGIC DIAGNOSIS	1473
Clinic of Drs. Solomon Strouse and Phil A Daly <i>Chicago Lying-In Hospital for</i> <i>Northwestern University</i>	
DIABETES AND PREGNANCY	1491
Clinic of Dr Ralph C Hamill <i>Wesley Memorial Hospital</i>	
DISABILITY DANGERS OF DISEASE	1499
Clinic of Dr Harold A Bachmann <i>From the Cardiac Clinic of the Children's Memorial Hospital</i>	
THE PREVENTION OF HEART DISEASE IN CHILDHOOD	1531

	PAGE
Clinic of Dr William H Holmes Wesley Memorial Hospital	
CASES ILLUSTRATING THE SYMPTOMATOLOGY OF BRAIN-STEM LESIONS AND OF CHRONIC SUBDURAL HEMORRHAGE	1541
Clinic of Dr Sidney Strauss, Cook County and Michael Reese Hospitals	
DISEASES OF THE BLOOD WITH SPECIAL REFERENCE TO HEMORRHAGES	1563
Clinic of Drs Julius H Hess and Philip Rosenthal, Sarah Morris Children's Hospital of the Michael Reese Hospital	
GASTRO INTESTINAL HEMORRHAGE IN CHILDREN	1577
Clinic of Dr Jacob Meyer Michael Reese Hospital	
I ORTHOSTATIC ALBUMINURIA	1591
II ACUTE GLOMERULAR NEPHRITIS	1593
III CHRONIC GLOMERULAR NEPHRITIS	1595
IV ACUTE YELLOW ATROPHY	1598
Clinic of Dr Harry A Singer Cook County Hospital	
SOME COMPLICATED AND UNCOMMON GASTRIC LESIONS	1601
Clinic of Drs William A Brants and E P Wolf-Blitzsten, Michael Reese Hospital	
CLINICALLY DORMANT CARCINOMA AND THE IMPORTANCE OF A COMPLETE PHYSICAL EX- AMINATION PAROXYSMAL TACHYCARDIA	1627
<hr/>	
INDEX TO VOLUME 9	1637

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